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ORIGINAL ARTICLES

CHEMICAL STUDIES OF THE BLOOD IN PNEUMONIA

I. A Method of Constructing an Alignment Diagram to Represent Changes in the Gaseous Composition of the Blood and Changes in the Electrolyte Concentration of the Plasma.*

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SINCE the work of Barcroft[1] in 1913 on the oxygen dissociation curve of the blood there has been a rapid development of our knowledge concerning the respiratory mechanism. The early work of Barcroft, of Bohr[2] and of Krogh[3] has been followed by the investigations of Christiansen, Douglas and Haldane[4], Hasselbalch[5], Parsons[6], Peters[7], Fridericia[8], L. J. Henderson[9], Y. Henderson[10], Lundsgaard[11], Van Slyke[12] and others all contributing to the general problem of the physico-chemical system of the blood. Y. Henderson and Haggard[13] in 1919 first attempted to bring together, graphically as many of the factors as were then accurately known in the form of their carbon dioxide diagram of the blood. At that time, however, the effect of oxygenation of the blood upon the carbon dioxide exchange was not fully appreciated and it remained for L. J. Henderson to describe the mechanism by which carbon dioxide is driven off in the process of oxidation and to demonstrate how the several factors,—hydrogen-ion concentration, percentage oxygen saturation, oxygen and carbon dioxide tensions, bicarbonate and chloride concentrations,—could be grouped as interrelated factors of a physico-chemical system and represented graphically by means of an alignment diagram or nomogram.

More recently, Bock, Field and Adair[14] have determined with great accuracy the carbon dioxide dissociation curves of the blood laying part of the foundation for the paper by Henderson, Bock, Field and Stoddard[15]. In this latter publication additional data are presented and from a consideration of these, and the data presented by Van Slyke, Wu and McLean[16] a nomogram of the blood is constructed showing the changes which may occur at all parts of the arterio-venous cycle.

It might seem superfluous, after these publications, to present still another method of constructing alignment diagrams of the blood. Our reason for so doing is that we believe that the methods here detailed are in some respects simpler than those hitherto described and represent variations which may have particular usefulness in the study of special conditions. Moreover, one of the diagrams here presented retains a desirable feature of the original Henderson-Haggard diagram, namely, the shape of the carbon dioxide dissociation curve which could be apprehended only vaguely from inspection of the alignment diagrams hitherto published.

PROCEDURE

Analyses of blood for carbon dioxide and oxygen were made with the apparatus and according to the method described by Van Slyke[17]. The sodium chloride content of the blood was determined by the method of Smith[18]. Since the analyses here reported were carried out a very much more accurate method for blood gas analysis has been described by Van Slyke[19] wherein an apparatus is used in which the volume of gas is maintained constant. Although our data are undoubtedly less accurate than those presented in very recent papers and though many discrepancies are obvious we believe, nevertheless, that the values obtained are sufficiently accurate to show the essential truth of the propositions which we wish to demonstrate. Following is the general description of the plan followed in collecting the data for determining the fundamental relationships on which the nomogram is based.

Specimens of oxalated blood were nearly deprived of oxygen by subjecting them to an atmosphere of carbon dioxide. This procedure was adopted because in practice it is very difficult to secure consistent results by starting with fully oxygenated blood and then simply lowering the oxygen tension of the atmosphere. It is necessary to start with reduced blood and, maintaining the carbon dioxide tension constant, gradually to increase the tension of oxygen to which the blood is exposed. Accordingly, specimens of reduced blood in rotating cylinders were exposed to certain tensions of carbon dioxide and oxygen for a period of twenty minutes at 37°C. The tensions of carbon dioxide used were such as to yield data showing the influence of increasing oxygen tension on blood maintained at a constant carbon dioxide tension. The actual tensions maintained are stated in Table I.

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*From the Department of Pediatrics of the Harvard Medical School and the Pediatric Service and the Blood Laboratory of the Boston City Hospital. This is publication No. 57 of a series of studies on metabolism from the Harvard Medical School and allied hospitals. The expenses of this research were in part defrayed by a grant from the Proctor Fund of Harvard University for the study of chronic disease.

TABLE I

OXYGEN DISSOCIATION OF HEMOGLOBIN
(Percent Oxyhemoglobin)

	Carbon Dioxide Tension (m.m.Hg)			
Oxygen Tension (m.m.Hg)	20	40	70	140
10	26.3	20.8	24.4	22.9
20	48.5			20.5
30	82.1	65.6	48.3	40.6
40	89.3	75.3		
50	90.4	85.1	72.6	70.5
60	88.6			
70	94.5	89.4	86.0	79.0
80				
90	93.1		90.1	92.0
100	94.1	93.5		
110	94.4		91.4	91.3
130				92.9
145			94.3	
151		95.7		
155	96.6			

In particular, a given sample of freshly drawn venous blood, after reduction, was divided into three parts. One of these was exposed to atmospheric air. A second part of the sample was exposed simultaneously to an atmosphere in which the tension of carbon dioxide was, for example, 20 mm. Hg and the oxygen tension 10 mm. Hg. At the same time the third part of the sample was exposed to an atmosphere in which the tension of carbon dioxide was 20 mm. Hg and the tension of oxygen 155 mm. Hg.* After equilibration the first sample was analyzed for its content of sodium chloride and of oxygen. The latter represented the oxygen combining capacity of the blood. A part of the second part of the sample was analyzed for its contents of oxygen and carbon dioxide as well as sodium chloride. Another part of this second specimen was centrifugalized under oil and the plasma so obtained analyzed for its contents of sodium chloride and carbon dioxide. Similarly, an analysis was made of the third part of the original sample of reduced blood. From these data the oxygen dissociation curves (Chart I) were drawn as well as the graphs representing the percentage change in carbon dioxide content of the plasma (Chart II) and the whole blood (Chart III) and the percentage change in the sodium chloride content of the plasma (Chart IV) with changes in the percentage saturation of the whole blood with oxygen.

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*If the carbon dioxide tension were 40 mm. Hg, the oxygen tension was made 145 mm. Hg, and if the carbon dioxide tension were 70 mm. Hg, the oxygen tension was made 145 mm. Hg. Finally, if the carbon dioxide tension were 140 mm. Hg, the oxygen tension was made 130 mm. Hg. This variation was introduced in order to make the values in the fundamental series of curves correspond with those obtained in the study of a particular case. In obtaining the carbon dioxide dissociation curve of the blood of a given patient it was our practice to subject the blood to tensions of carbon dioxide of 20, 40 and 70 mm. Hg by allowing a known volume of carbon dioxide to displace an equal volume of atmospheric air from the tonometer. This procedure caused, of course, a diminution of the oxygen tension in the air of the tonometer and at a barometric pressure of 760 mm. Hg the oxygen tensions would have values equal to those assigned above.

Oxygen Dissociation At Constant Carbon Dioxide Tensions.
Chart No I

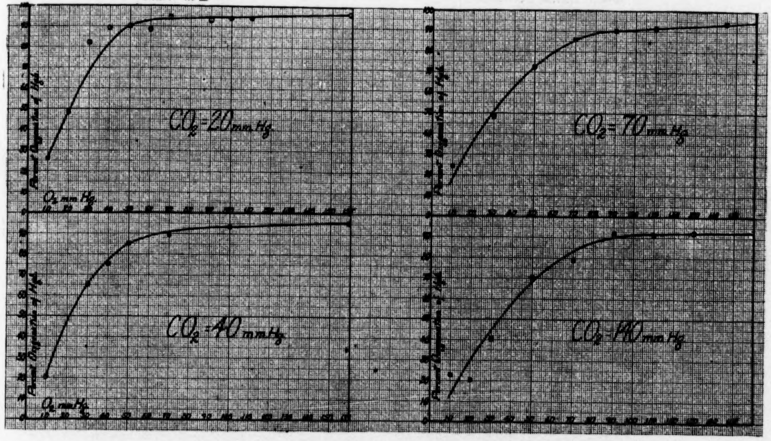


TABLE II

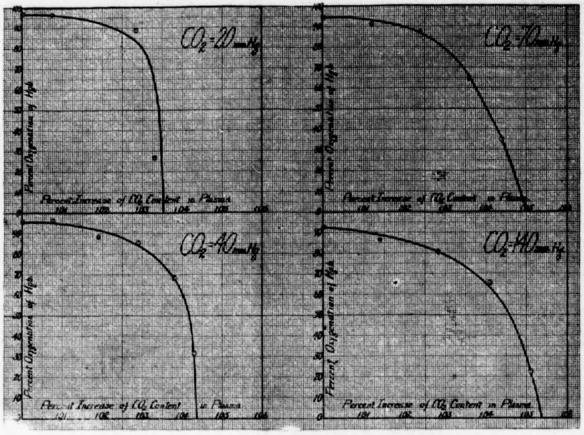
Percentage Increase in Plasma CO_2 Content with Decrease in Percentage $HgbO_2$ at CO_2 Tensions of 20, 40, 70, and 140 m.m. Hg.

20 m.m. CO_2		40 m.m. CO_2		70 m.m. CO_2		140 m.m. CO_2	
percentage $HgbO_2$	percentage CO_2 increase	percentage $HgbO_2$	percentage CO_2 increase	percentage $HgbO_2$	percentage CO_2 increase	percentage $HgbO_2$	percentage CO_2 increase
26.5	3.3	31.9	4.3	35.0	4.4	22.5	5.1
88.8	2.9	68.1	3.8	64.7	3.6	65.6	4.1
96.5	0.8	85.4	2.9	87.5	2.4	80.6	2.9
96.6	0.0	88.4	1.9	91.4	1.2	86.7	1.4
		96.4	0.8	94.3	0.0	92.9	0.0
		95.7	0.0				

Several blood specimens were subjected to each of the tensions of carbon dioxide and oxygen indicated in Table I, the number varying from three to seventeen and depending upon the closeness with which the analytical results agreed with one another. Inasmuch as several hundred analyses were made the results from individual specimens are not here presented but the mean values are shown in Tables I, II, III and IV.

Percentage Increase Of Plasma CO_2 Content Corresponding To The Decrease Of Percent O_2 Saturation Of The Whole Blood At Constant CO_2 Tensions.

Chart II.



In Table I are recorded the mean values for the percentage saturation of the blood with oxygen at fixed tensions of carbon dioxide and at increasing oxygen tensions. From these data the oxygen dissociation curves of Chart I were constructed. In Table II the mean percentage increase of the carbon dioxide content of the plasma corresponding to a given mean percentage decrease in the percentage saturation of the blood with oxygen at a given carbon dioxide tension is shown. Similarly, in Table III the mean percentage increase in the carbon dioxide content of the whole blood corresponding to a given mean percentage decrease in the percentage saturation of the blood with oxygen at a fixed carbon dioxide tension is shown. These results are represented graphically in Charts II and III. In each instance, for the purpose of calculating percentage changes the value of 100 was assigned to the carbon dioxide

TABLE III

PERCENTAGE INCREASE IN WHOLE BLOOD CO_2 CONTENT WITH DECREASE IN PERCENTAGE $HgbO_2$ AT CO_2 TENSIONS OF 20, 40, 70, AND 140 M.M. HG.

20 m.m. CO_2		40 m.m. CO_2		70 m.m. CO_2		140 m.m. CO_2	
Per-centage $HgbO_2$	per-centage CO_2 increase	per-centage $HgbO_2$	per-centage CO_2 increase	per-centage $HgbO_2$	per-centage CO_2 increase	per-centage $HgbO_2$	per-centage CO_2 increase
28.3	4.9	28.5	5.3	33.1	6.5	27.9	7.2
88.5	2.8	85.7	2.8	82.3	4.0	74.3	4.8
89.4	1.3	92.5	1.1	85.9	1.4	92.9	0.0
96.6	0.0	95.7	0.0	88.0	2.6		
				91.6	0.4		
				94.3	0.0		

content of the blood exposed to a given tension of carbon dioxide and the maximal oxygen tension used at that tension of carbon dioxide (third part of original sample of blood).

Percentage Increase In Whole Blood CO_2 Content Corresponding To The Decrease Of Percent O_2 Saturation Of The Whole Blood At Constant CO_2 Tensions
Chart III.

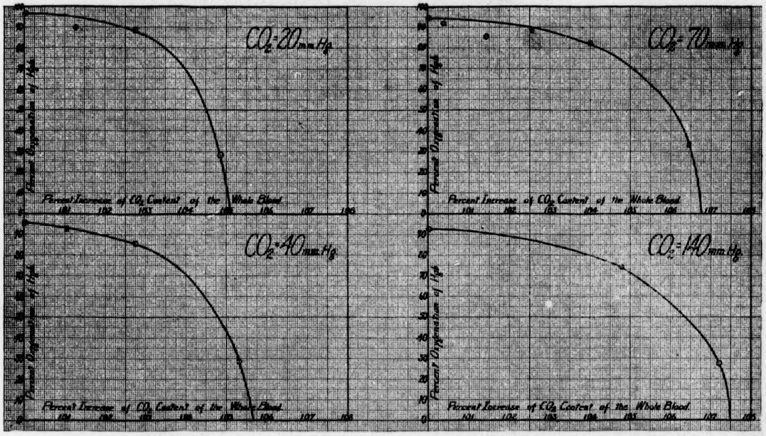


TABLE IV

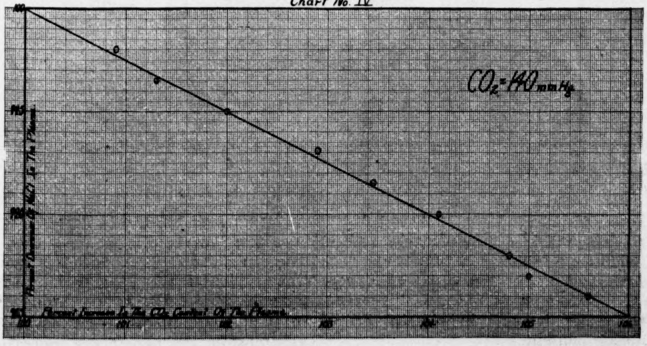
PERCENTAGE DECREASE IN PLASMA NACL WITH PERCENTAGE INCREASE IN PLASMA CO_2

Percentage CO_2 increase	Percentage NaCl decrease
0.9	0.2
1.3	0.3
2.0	0.5
2.9	0.7
3.5	0.8
4.1	1.0
4.8	1.2
5.0	1.3
5.6	1.4

In Table IV are shown the percentage changes in the sodium chloride content of the plasma corresponding to percentage changes in the carbon dioxide content of the plasma and these relationships are represented in the graph of Chart IV.

Since the percentage increase in the carbon dioxide content of the plasma corresponding to a given diminution of oxygen saturation of the whole blood has been determined (Table II) and since the relationship between the percentage increase in the plasma carbon dioxide content and the percentage decrease in the plasma sodium chloride content has also been computed (Table IV), it is possible so to align these values that the relation between percentage changes in the plasma content of sodium chlor-

Percent Decrease Of NaCl In The Plasma Corresponding To The Percent Increase Of CO_2 Content Of The Plasma.
Chart No. IV



ide and changes in the percentage saturation of the blood with oxygen can be evaluated (Table V). It is now possible to represent simultaneously the percentage saturation of the whole blood with oxygen at a given tension of carbon dioxide as a function of the following variables:

(a) Oxygen Tension.
(b) Percentage increase in plasma carbon dioxide content.
(c) Percentage increase in whole blood carbon dioxide content.
(d) Percentage decrease in plasma sodium chloride content.

TABLE V

PERCENTAGE DECREASE IN PLASMA NaCl COMPARED WITH PERCENTAGE OXYGENATION OF WHOLE BLOOD OBTAINED BY COMBINING TABLES II AND IV

CO_2 tension m.m. Hg	percentage increase in Plasma CO_2	percentage decrease in Plasma NaCl	percentage $HgbO_2$
20	0.0	0.0	96.6
	1.0	0.25	95.3
	2.0	0.50	85.7
	3.0	0.75	61.3
	3.5	0.88	34.8
	3.78	0.946	0.0
40	0.0	0.0	95.7
	1.0	0.25	93.4
	2.0	0.50	87.6
	3.0	0.75	75.3
	4.0	1.00	48.3
	4.5	1.12	13.8
	4.6	1.15	0.0
70	0.0	0.0	94.3
	1.0	0.25	93.5
	2.0	0.50	90.4
	3.0	0.75	80.4
	4.0	1.00	54.7
	4.5	1.12	27.3
	4.74	1.19	0.0
140	0.0	0.0	92.9
	1.0	0.25	90.0
	2.0	0.50	85.9
	3.0	0.75	79.5
	4.0	1.00	67.3
	4.5	1.12	56.5
	5.0	1.25	36.8
	5.35	1.34	0.0

The representation of these simultaneous relationships are shown in the graphs of Charts V, VI, VII and VIII. From these graphs, by interpolating values at short intervals, the results recorded in Tables VI, VII, VIII and IX were obtained. From these values, Chart IX was constructed. This chart, or nomogram, is really merely a rectification of the graphs of Charts V, VI, VII and VIII. From it, there may be readily ascertained, for a given percentage saturation of the blood at a given tension of carbon dioxide, the change in the oxygen tension, the percentage increase in the car-

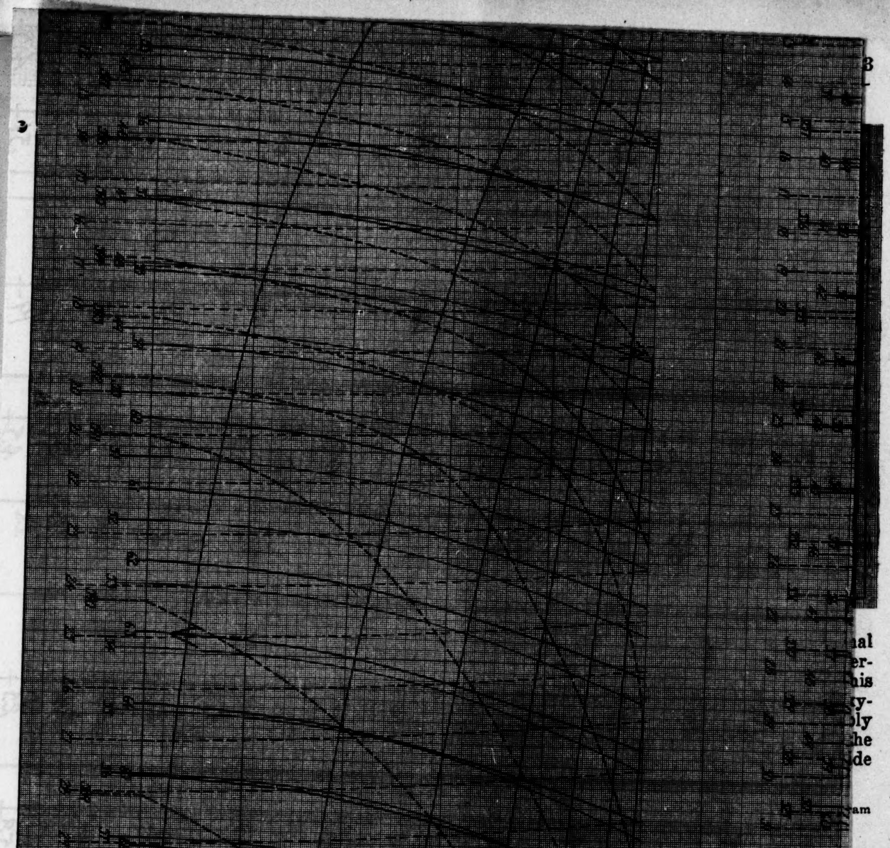


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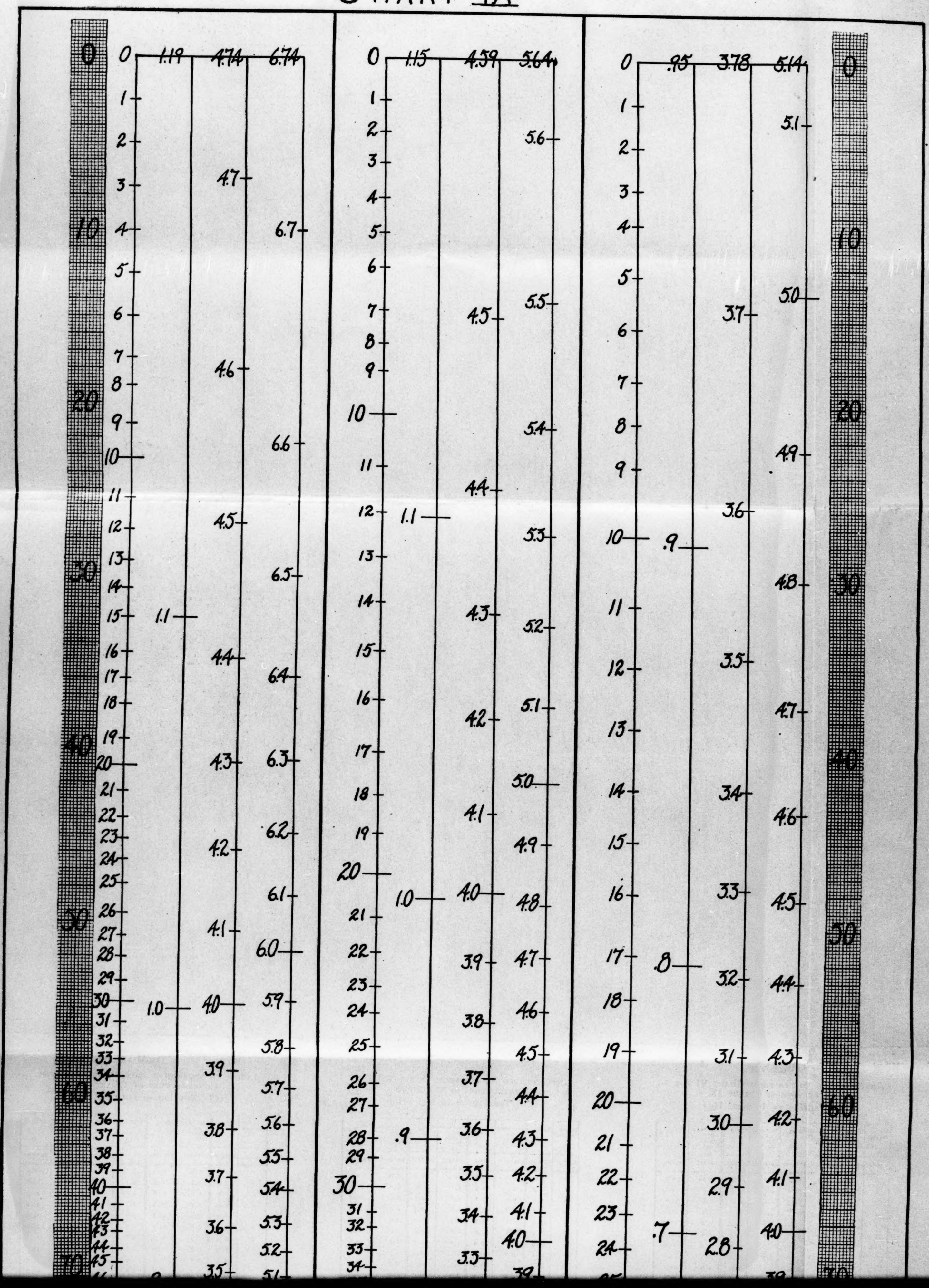
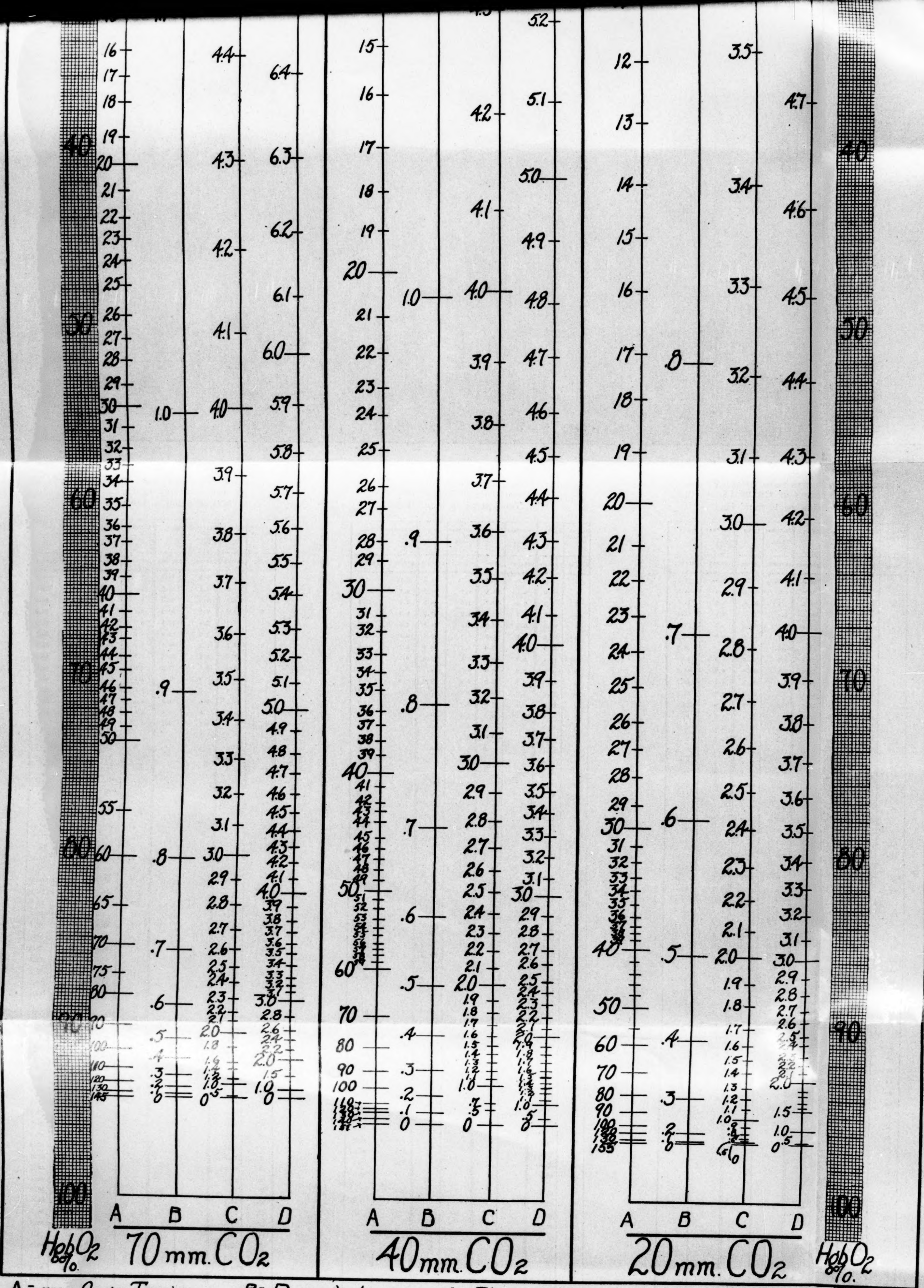
CHART IX





A = mm. Oxygen Tension B = Percent decrease of Plasma NaCl
C = Percent increase of Plasma CO2. D = Percent increase of Whole Blood CO2.

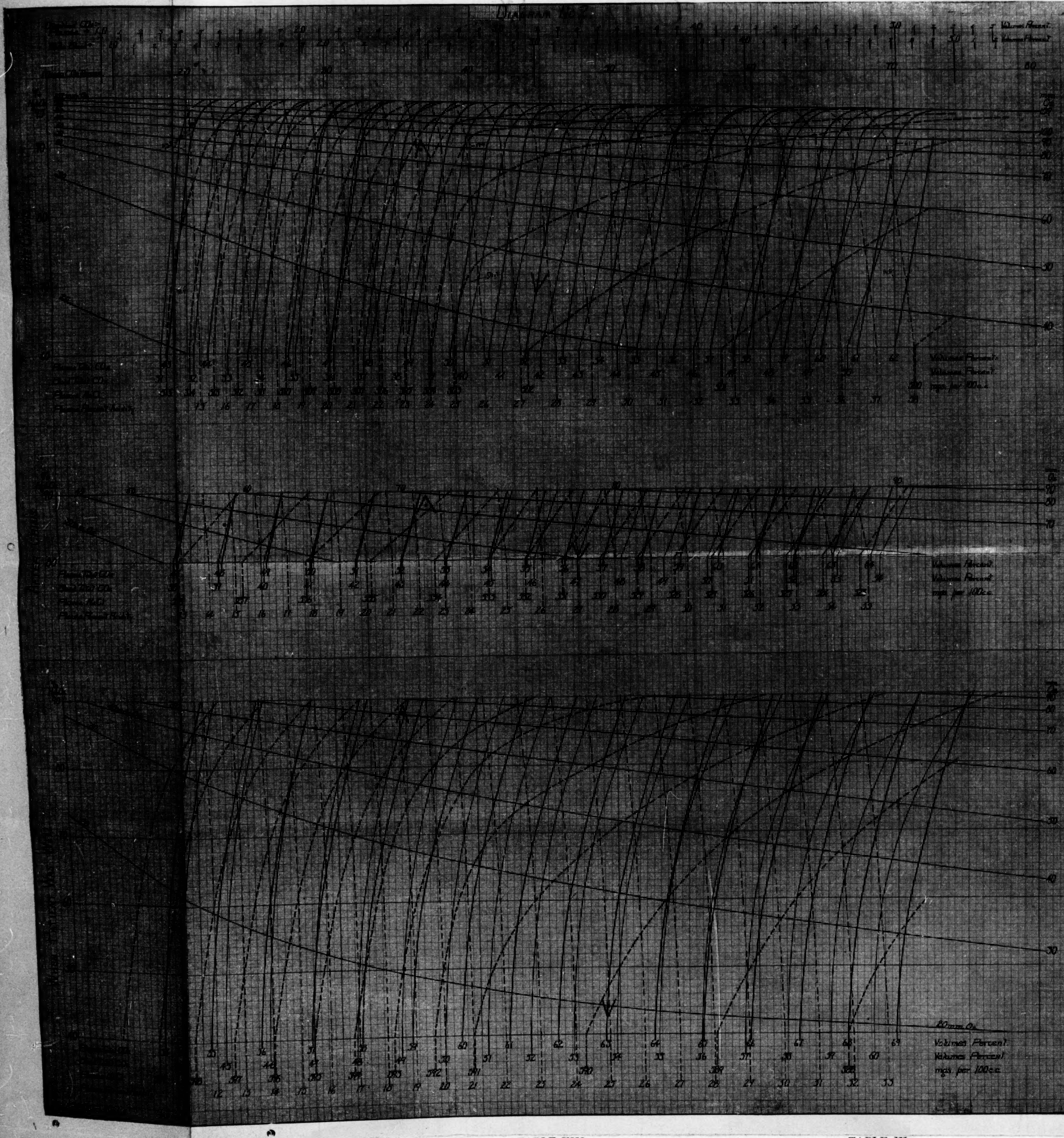


TABLE VII

Interpolated Points from Chart VI for Construction of Chart IX (CO$_2$ Tension = 40 m.m. Hg)

Oxygen Tension	HgbO$_2$	Decrease of Plasma NaCl	HgbO$_2$	Increase of Plasma CO$_2$	HgbO$_2$	Increase of Whole Blood CO$_2$	HgbO$_2$
m.m.Hg	percent	percent	percent	percent	percent	percent	percent
0	0.0	0.0	95.7	4.59	0.0	5.64	0.0
1	2.4	0.1	95.0	4.5	14.9	5.6	4.5
2	4.8	0.2	94.0	4.4	24.8	5.5	14.0
3	6.0	0.3	93.5	4.3	32.0	5.4	21.3
4	8.0	0.4	90.4	4.2	38.0	5.3	27.5
5	10.0	0.5	87.5	4.1	43.5	5.2	32.7
6	12.0	0.6	83.6	4.0	48.0	5.1	37.3
7	14.5	0.7	78.6	3.9	52.0	5.0	41.7
8	16.4	0.8	71.6	3.8	55.5	4.9	45.2
9	18.0	0.9	63.4	3.7	58.8	4.8	48.7
10	20.5	1.0	48.4	3.6	61.8	4.7	51.7
11	23.5	1.1	25.5	3.5	64.5	4.6	54.8
12	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	4.5	57.3

TABLE VIII

Interpolated Points from Chart VII for Construction of Chart IX (CO$_2$ Tension = 70 m.m. Hg)

Oxygen Tension	HgbO$_2$	Decrease of Plasma NaCl	HgbO$_2$	Increase of Plasma CO$_2$	HgbO$_2$	Increase of Whole Blood CO$_2$	HgbO$_2$
m.m.Hg	percent	percent	percent	percent	percent	percent	percent
0	0.0	0.0	94.3	4.74	0.0	6.74	0.0
1	2.5	0.1	94.1	4.7	7.0	6.7	10.0
2	5.0	0.2	93.7	4.6	18.0	6.6	23.3
3	7.5	0.3	93.0	4.5	27.0	6.5	30.0
4	10.0	0.4	92.0	4.4	34.8	6.4	35.8
5	12.5	0.5	90.8	4.3	40.8	6.3	40.6
6	15.0	0.6	88.6	4.2	45.8	6.2	44.8
7	17.4	0.7	85.7	4.1	50.5	6.1	48.4
8	19.0	0.8	80.5	4.0	54.7	6.0	51.6
9	21.2	0.9	71.0	3.9	58.6	5.9	54.5
10	23.2	1.0	55.0	3.8	62.0	5.8	57.2
11	25.5	1.1	32.5	3.7	64.8	5.7	59.6
12	27.4	1.19	0.0	3.6	67.7	5.6	61.7

TABLE IX

Interpolated Points from Chart VIII for Construction of Chart IX (CO$_2$ Tension = 140 m.m. Hg)

Oxygen Tension	HgbO$_2$	Decrease of Plasma NaCl	HgbO$_2$	Increase of Plasma CO$_2$	HgbO$_2$	Increase of Whole Blood CO$_2$	HgbO$_2$
m.m.Hg	percent	percent	percent	percent	percent	percent	percent
0	0.0	0.0	93.9	5.34	0.0	7.5	0.0
1	1.0	0.1	93.0	5.3	10.5	7.4	13.0
2	2.5	0.2	91.0	5.2	22.5	7.3	20.6
3	4.8	0.3	90.0	5.1	30.5	7.2	26.8
4	7.0	0.4	88.3	5.0	36.8	7.1	31.8
5	9.2	0.5	86.4	4.9	42.0	7.0	36.0
6	12.0	0.6	84.0	4.8	46.9	6.9	39.5
7	14.4	0.7	80.8	4.7	50.3	6.8	42.6
8	16.5	0.8	77.0	4.6	53.6	6.7	45.5
9	19.0	0.9	72.4	4.5	56.4	6.6	48.2
10	21.6	1.0	66.7	4.4	58.8	6.5	50.5
11	24.0	1.1	59.0	4.3	61.2	6.4	52.8
12	26.5	1.2	48.4	4.2	63.3	6.3	54.8

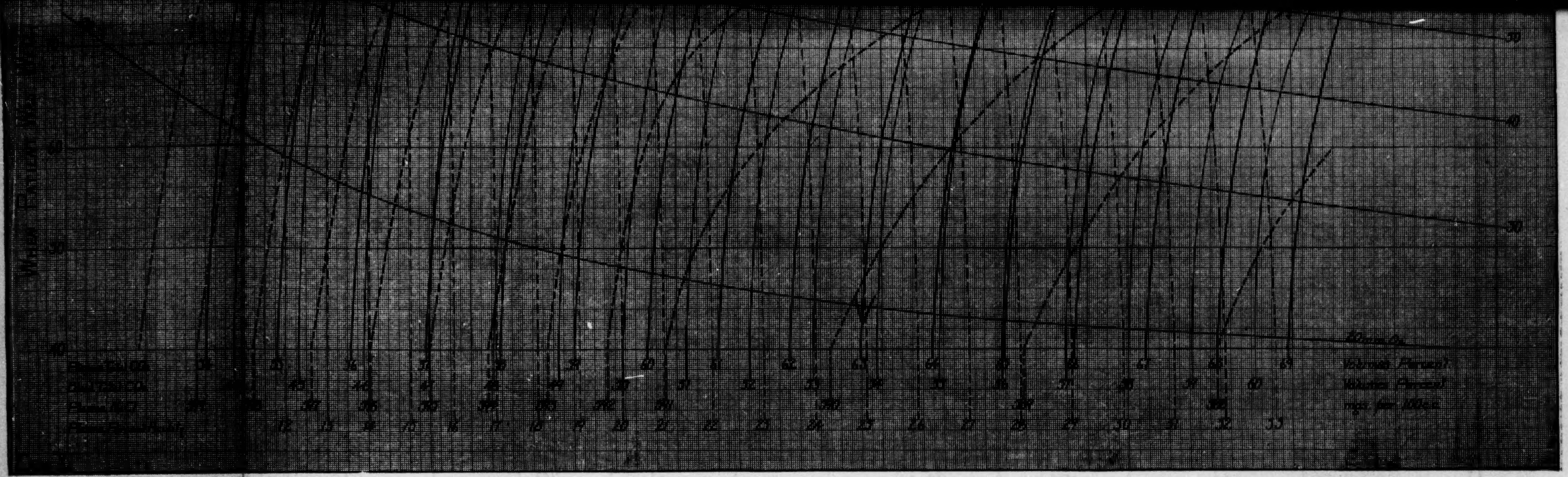


TABLE VII

INTERPOLATED POINTS FROM CHART VI FOR CONSTRUCTION OF CHART IX

(CO_2 Tension = 40 m.m. Hg)

Oxygen Tension	$HgbO_2$	Decrease of Plasma NaCl	$HgbO_2$	Increase of Plasma CO_2	$HgbO_2$	Increase of Whole Blood CO_2	$HgbO_2$
m.m.Hg	percent	percent	percent	percent	percent	percent	percent
0	0.0	0.0	95.7	4.59	0.0	5.64	0.0
1	2.4	0.1	95.0	4.5	14.9	5.6	4.5
2	4.2	0.2	94.0	4.4	24.8	5.5	14.0
3	6.0	0.3	92.5	4.3	32.0	5.4	21.3
4	8.0	0.4	90.4	4.2	38.0	5.3	27.5
5	10.0	0.5	87.5	4.1	43.5	5.2	32.7
6	12.0	0.6	83.6	4.0	48.0	5.1	37.3
7	14.5	0.7	78.6	3.9	52.0	5.0	41.7
8	16.4	0.8	71.6	3.8	55.5	4.9	45.2
9	18.0	0.9	63.4	3.7	58.8	4.8	48.7
10	20.5	1.0	48.4	3.6	61.8	4.7	51.7
11	23.5	1.1	26.5	3.5	64.5	4.6	54.8
12	26.3	1.15	0.0	3.4	66.8	4.5	57.3
13	28.5			3.3	69.2	4.4	59.8
14	31.4			3.2	71.2	4.3	62.3
15	34.2			3.1	73.2	4.2	64.4
16	37.0			3.0	74.9	4.1	66.5
17	40.0			2.9	76.6	4.0	68.2
18	42.5			2.8	78.2	3.9	70.2
19	44.7			2.7	79.7	3.8	72.0
20	47.0			2.6	81.0	3.7	73.5
21	49.5			2.5	82.2	3.6	75.0
22	51.5			2.4	83.4	3.5	76.5
23	53.5			2.3	84.5	3.4	77.7
24	55.0			2.2	85.5	3.3	79.0
25	57.0			2.1	86.5	3.2	80.2
26	59.2			2.0	87.4	3.1	81.4
27	60.5			1.9	88.3	3.0	82.4
28	62.4			1.8	89.0	2.9	83.5
29	63.5			1.7	89.7	2.8	84.5
30	65.2			1.6	90.4	2.7	85.4
31	66.5			1.5	91.0	2.6	86.3
32	67.5			1.4	91.5	2.5	87.2
33	68.8			1.3	92.0	2.4	87.5
34	69.9			1.2	92.5	2.3	88.5
35	70.8			1.1	93.0	2.2	89.2
36	72.0			1.0	93.4	2.1	89.9
37	72.8			0.7	94.4	2.0	90.4
38	73.7			0.5	94.8	1.9	91.0
39	74.5			0.0	95.7	1.8	91.5
40	75.5					1.7	92.0
41	76.3					1.6	92.5
42	77.2					1.4	93.2
43	77.7					1.2	93.8
44	78.3					1.1	94.2
45	79.2					1.0	94.5
46	79.8					0.5	95.3
47	80.4					0.0	95.7
48	81.0						
49	81.6						
50	82.2						
51	82.7						
52	83.2						
53	83.8						
54	84.3						
55	84.7						
56	85.2						
57	85.5						
58	85.8						
59	86.2						
60	86.6						
70	89.3						
80	91.2						
90	92.6						
100	93.6						
110	94.5						
120	94.8						
130	95.0						
140	95.4						
151	95.7						

TABLE VIII

INTERPOLATED POINTS FROM CHART VII FOR CONSTRUCTION OF CHART IX

(CO_2 Tension = 70 m.m. Hg)

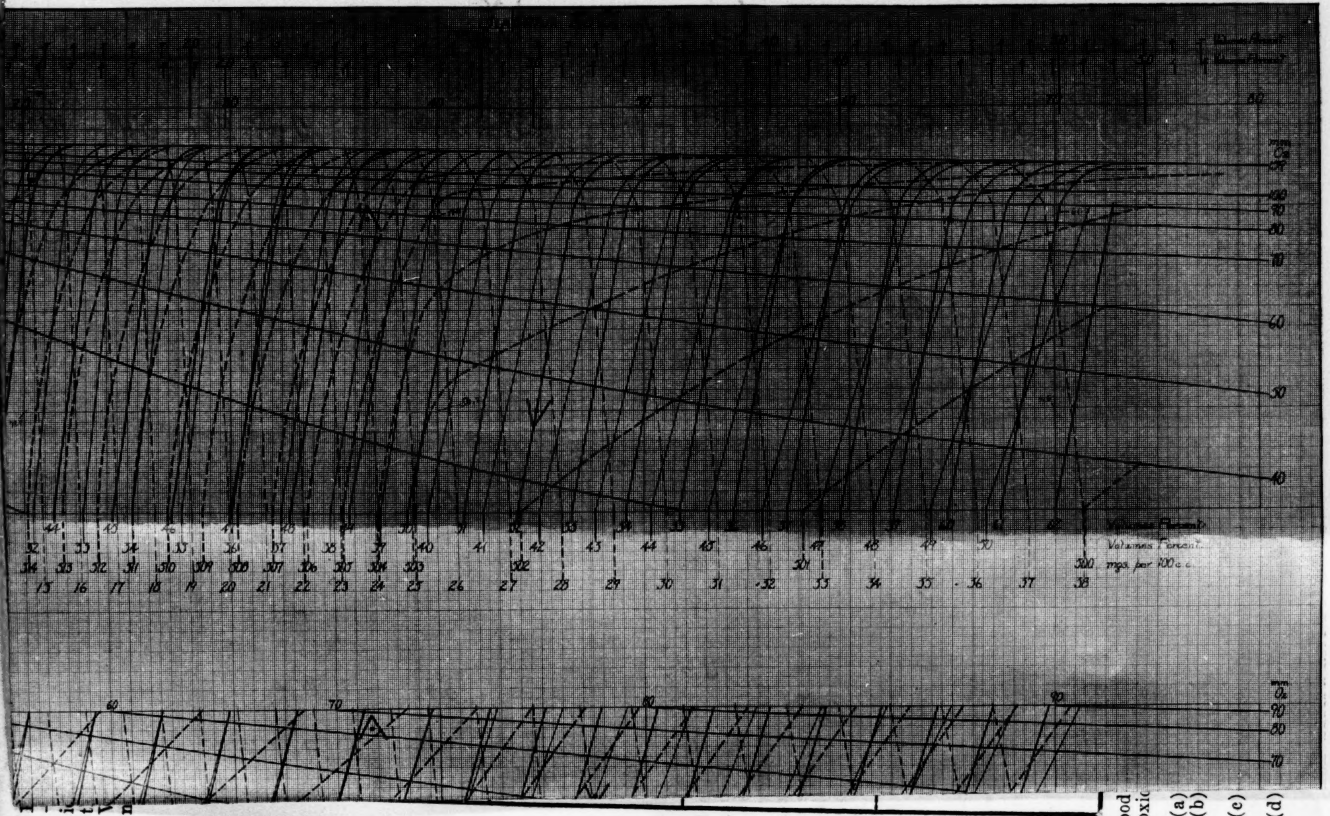
Oxygen Tension	$HgbO_2$	Decrease of Plasma NaCl	$HgbO_2$	Increase of Plasma CO_2	$HgbO_2$	Increase of Whole Blood CO_2	$HgbO_2$
m.m.Hg	percent	percent	percent	percent	percent	percent	percent
0	0.0	0.0	94.3	4.74	0.0	6.74	0.0
1	2.5	0.1	94.1	4.7	7.0	6.7	10.0
2	5.0	0.2	93.7	4.6	18.0	6.6	22.3
3	7.5	0.3	93.0	4.5	27.0	6.5	30.0
4	10.0	0.4	92.0	4.4	34.8	6.4	35.8
5	12.5	0.5	90.8	4.3	40.8	6.3	40.6
6	15.0	0.6	88.6	4.2	45.8	6.2	44.8
7	17.4	0.7	85.7	4.1	50.5	6.1	48.4
8	19.0	0.8	80.5	4.0	54.7	6.0	51.6
9	21.2	0.9	71.0	3.9	58.6	5.9	54.5
10	23.2	1.0	55.0	3.8	62.0	5.8	57.2
11	25.5	1.1	32.5	3.7	64.8	5.7	59.6
12	27.4	1.19	0.0	3.6	67.7	5.6	61.7
13	29.2			3.5	70.3	5.5	63.7
14	30.8			3.4	72.6	5.4	65.5
15	32.5			3.3	74.8	5.3	67.4
16	34.5			3.2	76.8	5.2	69.0
17	36.0			3.1	78.6	5.1	70.5
18	37.5			3.0	80.3	5.0	72.0
19	39.5			2.9	81.7	4.9	73.2
20	41.0			2.8	83.1	4.8	74.5
21	42.5			2.7	84.5	4.7	75.6
22	44.0			2.6	85.5	4.6	76.8
23	45.2			2.5	86.5	4.5	77.9
24	46.4			2.4	87.5	4.4	78.9
25	47.8			2.3	88.4	4.3	79.8
26	49.5			2.2	89.2	4.2	80.7
27	50.8			2.1	89.7	4.1	81.6
28	52.0			2.0	90.4	4.0	82.4
29	53.4			1.9	90.8	3.9	83.2
30	54.6			1.8	91.3	3.8	84.0
31	55.8			1.7	91.7	3.7	84.6
32	57.0			1.6	92.2	3.6	85.3
33	58.0			1.5	92.4	3.5	85.8
34	59.0			1.4	92.6	3.4	86.5
35	60.4			1.3	92.9	3.3	87.2
36	61.6			1.2	93.2	3.2	87.5
37	62.5			1.1	93.4	3.1	88.0
38	63.6			1.0	93.5	3.0	88.5
39	64.5			0.9	93.7	2.9	89.0
40	65.5			0.8	93.8	2.8	89.4
41	66.5			0.7	94.0	2.7	89.7
42	67.4			0.6	94.1	2.6	90.2
43	68.0			0.5	94.2	2.5	90.5
44	69.0			0.0	94.3	2.4	90.8
45	69.8					2.3	91.2
46	70.8					2.2	91.4
47	71.5					2.1	91.7
48	72.2					2.0	92.0
49	73.0					1.9	92.2
50	73.8					1.8	92.4
55	77.8					1.7	92.6
60	80.4					1.6	92.8
65	83.2					1.5	93.0
70	85.4					1.4	93.2
75	87.0					1.3	93.4
80	88.2					1.2	93.5
90	90.0					1.1	93.6
100	91.5					1.0	93.8
110	92.6					0.5	94.3
120	93.4					0.0	94.3
130	94.0						
145	94.3						

TABLE IX

INTERPOLATED POINTS FROM CHART VIII FOR CONSTRUCTION OF CHART IX

(CO_2 Tension = 140 m.m. Hg)

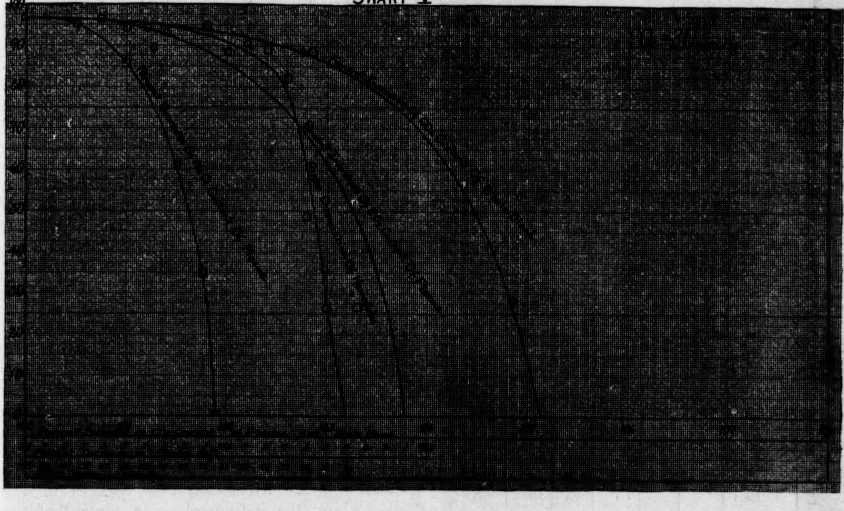
Oxygen Tension	$HgbO_2$	Decrease of Plasma NaCl	$HgbO_2$	Increase of Plasma CO_2	$HgbO_2$	Increase of Whole Blood CO_2	$HgbO_2$
m.m.Hg	percent	percent	percent	percent	percent	percent	percent
0	0.0	0.0	92.9	5.34	0.0	7.5	0.0
1	1.0	0.1	92.0	5.3	10.5	7.4	13.0
2	2.5	0.2	91.0	5.2	22.5	7.3	20.6
3	4.8	0.3	90.0	5.1	30.5	7.2	26.8
4	7.0	0.4	88.3	5.0	36.8	7.1	31.8
5	9.2	0.5	86.4	4.9	42.0	7.0	36.0
6	12.0	0.6	84.0	4.8	46.9	6.9	39.5
7	14.4	0.7	80.8	4.7	50.3	6.8	42.6
8	16.5	0.8	77.0	4.6	53.6	6.7	45.5
9	19.0	0.9	72.4	4.5	56.4	6.6	48.2
10	21.6	1.0	66.7	4.4	58.8	6.5	50.5
11	24.0	1.1	59.0	4.3	61.2	6.4	52.8
12	26.5	1.2	48.4	4.2	63.3	6.3	54.8
13	28.5	1.3	28.0	4.1	65.2	6.2	56.7
14	30.5	1.33	0.0	4.0	66.8	6.1	58.5
15	32.4			3.9	68.7	6.0	60.2
16	34.0			3.8	70.3	5.9	61.7
17	35.6			3.7	71.7	5.8	63.2
18	37.4			3.6	73.1	5.7	64.6
19	38.8			3.5	74.3	5.6	65.8
20	40.4			3.4	75.4	5.5	67.2
21	42.0			3.3	76.5	5.4	68.4
22	43.6			3.2	77.5	5.3	69.4
23	44.8			3.1	78.4	5.2	70.4
24	46.2			3.0	79.3	5.1	71.4
25	47.4			2.9	80.2	5.0	72.4
26	48.8			2.8	80.8	4.9	73.2
27	49.8			2.7	81.6	4.8	74.0
28	51.0			2.6	82.4	4.7	74.8
29	52.2			2.5	82.8	4.6	75.6
30	53.2			2.4	83.6	4.5	76.4
31	54.2			2.3	84.3	4.4	77.2
32	55.2			2.2	84.8	4.3	77.8
33	56.2			2.1	85.4	4.2	78.5
34	57.0			2.0	86.0	4.1	79.2
35	57.8			1.9	86.5	4.0	79.8
36	58.8			1.8	87.0	3.9	80.5
37	59.7			1.7	87.4	3.8	81.2
38	60.4			1.6	87.6	3.7	81.6
39	61.3			1.5	88.4	3.6	82.2
40	62.0			1.4	88.7	3.5	82.7
41	62.7			1.3	89.2	3.4	83.4
42	63.7			1.2	89.5	3.3	83.8
43	64.5			1.1	89.8	3.2	84.3
44	65.2			1.0	90.2	3.1	84.7
45	66.0			0.9	90.5	3.0	85.2
46	66.7			0.8	90.8	2.9	85.6
47	67.3					2.8	86.0
48	67.8					2.7	86.4
49	68.4					2.6	86.7
50	69.1					2.5	87.2
51	69.7					2.4	87.5
52	70.2					2.3	87.9
53	70.7					2.2	88.1
54	71.4					2.1	88.4
55	72.0					2.0	88.6
56	72.5					1.9	89.0
57	72.9					1.8	89.3
58	73.4					1.7	89.5
59	73.9					1.6	89.8
60	74.4					1.5	90.0
62	75.3					1.0	91.2
64	76.2					0.5	92.2
66	77.2					0.0	92.9
68	77.7						
70	78.5						
72	79.2						
74	79.9						
76	80.6						
78	81.2						
80	81.8						
82	82.3						
84	83.0						
86	83.7						
88	84.1						
90	84.5						
92	85.0						
94	85.4						
96	85.9						
98	86.3						
100	91.8						
110	92.0						
130	92.6						



blood
dioxi

(a)
(b)
(c)
(d)

CHART V



bon dioxide content of the whole blood and the plasma, and the percentage decrease in the sodium chloride content of the plasma relative to the values that any specimen of blood would yield by actual analysis when exposed to an atmosphere created by displacement of a sufficient volume of atmospheric air by carbon dioxide to produce the given tension of carbon dioxide and the given percentage saturation with oxygen. In a word, Chart IX is a computing machine for correcting the original carbon dioxide dissociation diagram of Henderson and Haggard.* But in addition to this the chart gives information concerning the oxygen tension to which the blood was presumably exposed and also permits a statement of the conditions under which the shift in the chloride ion content of the plasma occurs.

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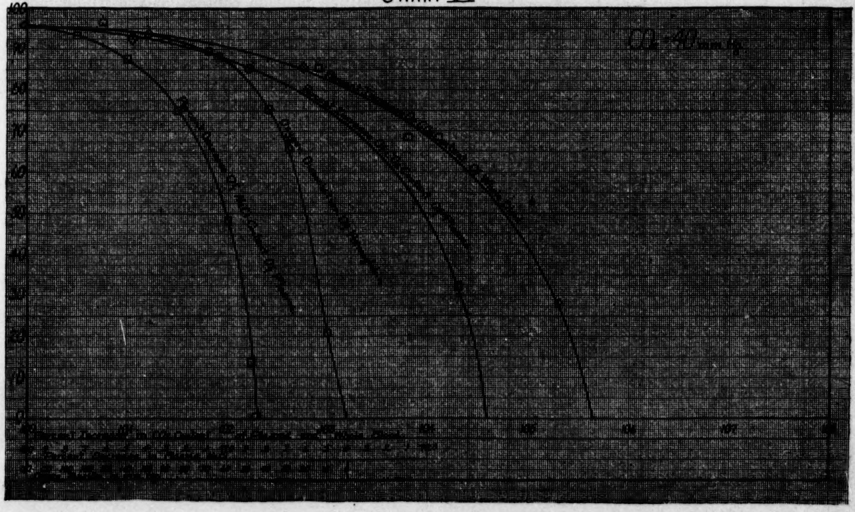
*Another method of correcting this carbon dioxide diagram has been described by Peters[7].

CHART VI



DETAILS OF CONSTRUCTION OF DIAGRAMS

The data thus far discussed may be used for constructing diagrams involving at least two different methods of alignment. In the first one to be described the rectangular ordinates represent the carbon dioxide content of the whole blood or plasma and the abscissae the tensions of carbon dioxide. Carbon dioxide dissociation curves corrected for both the arterial and venous percentage oxygen saturations of the blood are then drawn and on them placed respectively the arterial (A) and the venous (V) points. In the second form of diagram to be described the ordinates represent percentage oxygen saturation and the abscissae tensions of carbon dioxide. In the former type it is, practically, necessary to construct a separate chart for plasma and for whole blood; in the latter, both plasma and whole blood may be represented on a single chart. In the former only is it possible to exhibit the shape of the carbon dioxide dissociation curve.

TABLE VI

INTERPOLATED POINTS FROM CHART V FOR CONSTRUCTION OF CHART IX

(CO_2 Tension = 20 m.m. Hg)

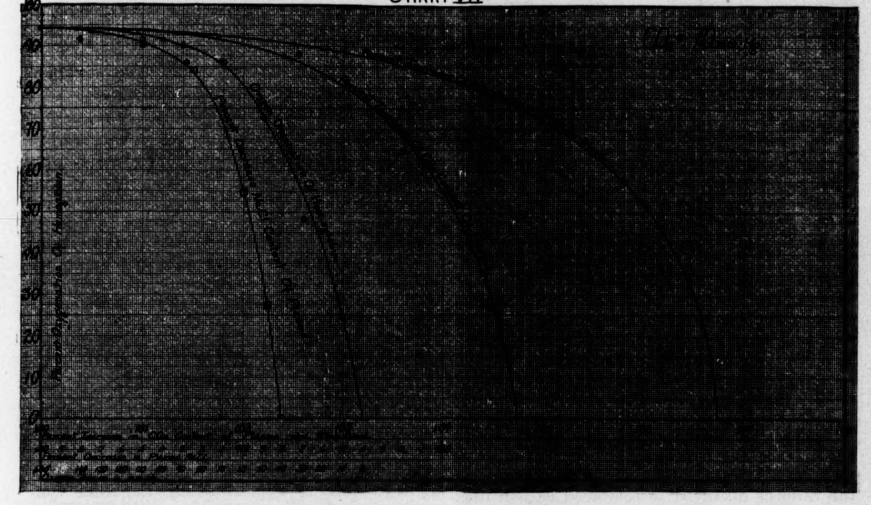
Oxygen Tension	HgbO$_2$	Decrease of Plasma NaCl	HgbO$_2$	Increase of Plasma CO$_2$	HgbO$_2$	Increase of Whole Blood CO$_2$	HgbO$_2$
m.m.Hg	percent	percent	percent	percent	percent	percent	percent
0	0.0	0.0	96.6	3.78	0.0	5.14	0.0
1	2.5	0.1	96.5	3.7	14.5	5.1	3.5
2	5.0	0.2	96.0	3.6	25.8	5.0	13.5
3	7.5	0.3	94.0	3.5	34.5	4.9	22.5
4	9.5	0.4	90.5	3.4	42.0	4.8	30.0
5	12.5	0.5	85.6	3.3	47.7	4.7	37.3
6	15.5	0.6	78.0	3.2	52.7	4.6	43.3
7	18.5	0.7	67.5	3.1	57.3	4.5	48.3
8	21.0	0.8	52.0	3.0	61.2	4.4	53.0
9	23.5	0.9	28.0	2.9	64.8	4.3	57.2
10	27.5	0.95	0.0	2.8	68.3	4.2	60.8
11	31.5			2.7	71.2	4.1	64.2
12	35.0			2.6	73.9	4.0	67.3
13	38.5			2.5	76.4	3.9	70.0
14	42.0			2.4	78.5	3.8	72.5
15	45.0			2.3	80.6	3.7	74.7
16	48.0			2.2	82.5	3.6	76.7
17	51.5			2.1	84.2	3.5	78.6
18	54.0			2.0	85.7	3.4	80.3
19	57.0			1.9	87.2	3.3	81.8
20	60.0			1.8	88.4	3.2	83.3
21	62.5			1.7	89.8	3.1	84.7
22	64.5			1.6	90.8	3.0	85.8
23	66.5			1.5	91.7	279	86.9
24	68.5			1.4	92.5	2.8	87.9
25	70.5			1.3	93.4	2.7	88.8
26	72.5			1.2	94.0	2.6	89.5
27	74.0			1.1	94.6	2.5	90.3
28	75.6			1.0	95.2	2.4	90.8
29	77.2			0.9	95.6	2.3	91.5
30	78.5			0.8	95.0	2.2	92.0
31	79.5			0.7	96.3	2.1	92.5
32	80.4			0.6	96.4	2.0	93.0
33	81.3			0.5	96.5	1.9	93.5
34	82.0			0.0	96.6	1.8	93.8
35	82.8					1.7	94.2
36	83.5					1.6	94.5
37	84.0					1.5	94.7
38	84.4					1.0	95.8
39	84.8					0.5	96.5
40	85.3					0.0	96.6
42	86.0						
44	86.7						
46	87.3						
48	87.9						
50	88.6						
55	89.8						
60	90.8						
70	92.5						
80	93.8						
90	94.8						
100	95.6						
120	96.0						
130	96.4						
150	96.6						
155	96.6						

It should be pointed out that a different diagram must be constructed for every individual whose blood is thus to be studied and on each occasion on which that individual is subjected to study. Whether one selects the first or the second method of alignment diagram now to be described the following data are necessary: (a) the carbon dioxide content of the whole blood and the carbon dioxide content of the plasma and the sodium chloride content of the plasma separated from the blood, equilibrated for twenty minutes at 37°C. in atmospheres in which the tensions of carbon dioxide and oxygen are 20, 40 and 70 mm. Hg. and 155, 151 and 145 mm. Hg respectively; (b) the oxygen capacity of the blood, the oxygen contents of the arterial and venous blood specimens taken into paraffine oil, the carbon dioxide contents of the whole blood and the plasma separated therefrom of both arterial and venous samples, the sodium chloride content of the whole blood and true plasma of both arterial and venous ples. From these data, then, with the aid of the diagram it is possible to determine the apparent oxygen and carbon dioxide tensions to which both arterial and venous blood specimens were exposed, the hydrogen ion concentrations of plasma and whole blood, both arterial and venous, the maximal shift in the chlorides from plasma to corpuscles as the blood passes from arterial to venous conditions.

TYPE I DIAGRAM

It is necessary at the outset to construct a skeleton diagram which may be used for any blood, this being essentially the one originally described by Henderson and Haggard. Here the ordinates represent content of carbon dioxide in volumes percent. and the abscissae tensions of carbon dioxide. It is advisable to use large sheets of millimeter squared paper, about 75x40 cm. and to allow at least 2.5 cm. per volumes percent. of carbon dioxide content and 0.5 cm. per mm. Hg of carbon dioxide tension.

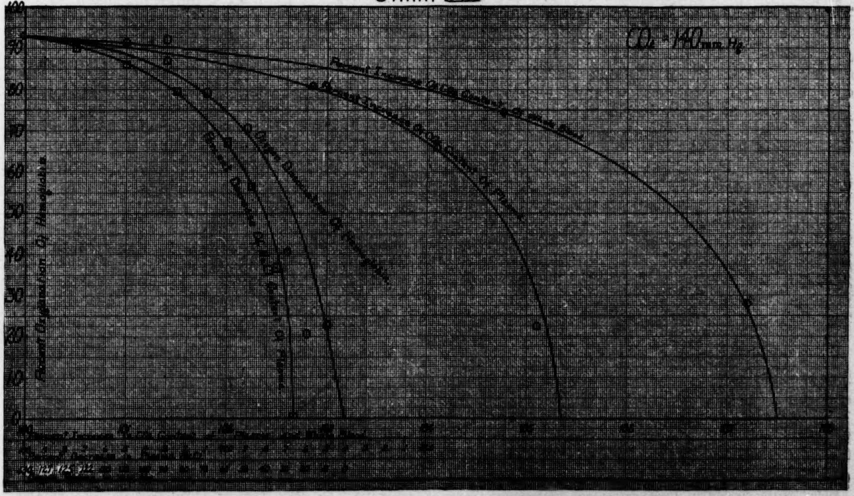
CHART VII



It will be found convenient in the final diagram to let the origin represent 20 mm. of carbon dioxide tension and about 35 volumes percent. of total carbon dioxide content in the case of whole blood, or 45 volumes percent. in the case of plasma, but in the preliminary construction the origin must represent zero for both carbon dioxide content and tension.

From the origin downwards below the axis of abscissae is drawn a straight line representing the solubility of carbon dioxide in whole blood or plasma at different tensions of the gas.

CHART VIII



Values for plotting this line are given by Bohr and are shown in the following table:

	Carbon	Dioxide	Tension	
	20 mm.Hg.	40 mm.Hg.	70 mm.Hg.	
Whole Blood	1.344	2.688	4.704	Content of dissolved carbon dioxide in volumes percent.
Plasma	1.424	2.848	4.984	

It is from this line and not from the axis of abscissae that all values for the total carbon dioxide content of the whole blood or plasma, as the case may be, are measured. Distance above the axis, then, represents combined carbon dioxide while distance below the axis represents dissolved carbon dioxide. For the sake of economy of space, after the carbon dioxide dissociation curves have been traced, this line may be deleted or placed above the base line.

When the distances corresponding to tensions of carbon dioxide and contents of carbon dioxide have been indicated and the solubility line drawn the isohydric lines or lines of equal hydrogen ion concentration may be constructed. Since the hydrogen ion concentration of the plasma, or whole blood, is proportional to the ratio of dissolved to combined carbon dioxide, any straight line drawn through the origin will be an isohydric line for such a line represents a fixed ratio of dissolved to combined carbon dioxide. If, therefore, the ratio of dissolved to combined carbon dioxide correspnding to any given hydrogen ion concentration be known it is possible to construct a series of isohydric lines which will include all possible physiologic values. For the purposes of this paper and those of this series which are to follow, it has seemed simpler to express hydrogen ion concentration in terms of percentage acidity rather than according to the logarithmic scale (pH). By percentage acidity we mean the ratio of the concentration of hydrogen ions in the plasma or whole blood to the concentration of hydrogen ions in pure water at 38°C. multiplied by 100.*

Now it is known that the hydrogen ion concentration of a solution containing carbonic acid in solution and sodium bicarbonate, for example, may be expressed by the following equation:

$$\text{(I)} \qquad (H^{\cdot}) = K\frac{(H_2CO_3)}{(HCO_3')}$$

But, inasmuch as in any solution containing both carbonic acid and sodium bicarbonate the relative degrees of dissociation of the two substances are such that, for the same mass of substance, the latter will yield over a million times the mass of bicarbonate ions as the former, it may be assumed for all practical purposes that the concentration of bicarbonate ions in such a solution is proportional to the concentration of sodium bicarbonate alone. Therefore, equation I may be written:

$$\text{(II)} \qquad (H^{\cdot}) = \frac{k_1(H_2CO_3)}{k_2(NaHCO_3)}$$

where k_1 and k_2 represent respectively the dissociation constants for carbonic acid and sodium bicarbonate. According to Michaelis and Rona the values for these constants in solutions containing approximately the same concentration of sodium chloride as the blood are as follows:

$k_1 = 4.40 \times 10^{-7}$ (Independent of temperature within physiologic ranges)

$k_2 = 0.605$ (Temperature gradient probably very slight)

Substituting these values in equation II we obtain the expression:

$$\text{(III)} \qquad (H^{\cdot}) = \frac{4.4 \times 10^{-7}(H_2CO_3)}{0.605(NaHCO_3)}$$

Now, as pointed out long ago by L. J. Henderson but not generally appreciated, pure water at 38 C. has a dissociation constant not of 1.0x10-14, as is often assumed, but of about 2.88x10-14. (Kohlrausch and Heydeweiller.) Whence, in pure water, at a temperature of 38 C.,

$$\text{(IV)} \qquad (H^{\cdot}) = 1.7 \times 10^{-7}$$

Accordingly, percentage acidity would be represented by the expression,

$$\frac{(H^{\cdot})}{1.7 \times 10^{-7}} \times 100$$

or, for the blood,

$$\text{Percentage Acidity } 38°\text{C.} = \frac{\dfrac{4.40 \times 10^{-7}(H_2CO_3)}{0.605(NaHCO_3)}}{1.7 \times 10^{-7}} \times 100$$

$$= \frac{4.40 \times 10^{-7}(H_2CO_3)}{0.605 \times 1.7 \times 10^{-7}(NaHCO_3)} \times 100$$

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*Notes. 1. More accurately, percentage acidity is the ratio of the concentration of hydrogen ions in plasma (or whole blood) to the concentration of hydrogen ions in pure water, both measured at the same temperature. This would require the computation of a different set of ratios for each temperature considered and this may be a necessary modification in studying the influence of temperature on the hydrogen ion concentration of the blood.

2. Whether the isohydric lines are designated percentage acidity lines, pH lines or hydrogen ion concentration lines, makes no difference regarding inferences that may be drawn concerning relative changes in the hydrogen ion concentration of the blood during the arterio-venous cycle, nor does it make any material difference whether the constants which we have employed in computing these ratios are changed by further investigations.

$$\text{or, (V)} \qquad = 427.8 \frac{(H_2CO_3)}{(NaHCO_3)}$$

In practice, the concentration of sodium bicarbonate (combined carbon dioxide) may be measured by volumes percent. and the concentration of dissolved carbon dioxide may be reckoned according to the following formula:

$$\text{Carbonic Acid (dissolved carbon dioxide) in volumes percent.} = \frac{0.541 \times 100TCO_2}{{}^{T}Bar} \qquad \text{(Bohr)}$$

where TCO_2 represents the partial pressure or tension of carbon dioxide and ^{T}Bar. represents the barometric pressure. Inasmuch as ordinary changes in the barometric pressure would produce very little change in the quantity of dissolved carbon dioxide, it is possible to write

$$\text{(VI)} \quad (H_2CO_3) \text{ in volumes percent. (Plasma)} = \frac{0.541 \times 100TCO_2}{760}$$

Combining equations V and VI we obtain the expression,

(VII) Percentage Acidity of Plasma at 38°C.

$$= \frac{TCO_2 \times 0.0712}{\text{Volumes Percent. Combined } CO_2} \times 427.8$$

$$= \frac{TCO_2}{\text{Volumes Percent. Combined } CO_2} \times 30.46$$

For whole blood, the solubility coefficient of carbon dioxide is 0.511 and equation VII becomes,

(VIII) Percentage Acidity for whole blood at 38°C.

$$= \frac{TCO_2}{\text{Volumes Percent. Combined } CO_2} \times 28.75$$

If, now, in the skeleton diagram a series of points be located on a horizontal line representing 30.46 volumes percent. of *combined* carbon dioxide at distances from the axis of ordinates represented by 10, 20, 30, 40 etc. mm. of carbon dioxide tension, and if through these points straight lines be drawn through the origin, then each of these will be an isohydric line whose percentage acidity will have the same numerical value as the carbon dioxide tension indicated by the vertical line intersecting the isohydric line at the level of the 30.46 volumes percent. line level. Another series of points may then be located on the horizontal line representing 2x30.46 or 60.92 volumes percent. of *combined* carbon dioxide. The value to be assigned to each of these lines will obviously be one half that of the corresponding line drawn through the lower level. In the same way, using equation VIII, percentage acidity lines may be constructed for the skeleton diagram for the whole blood.

TABLE XI

DATA OBTAINED FROM ANALYSES OF VENOUS BLOOD EXPOSED TO CARBON DIOXIDE TENSIONS OF 20, 40 AND 70 M.M. HG, THE CORRESPONDING OXYGEN TENSIONS BEING 155, 151 AND 145 M.M.

A.

CO_2 Tensions	Data from Analyses			Data from Analyses Corrected to 89 percent $HgbO_2$ with aid of Chart IX.			Data from Analyses Corrected to 70 percent $HgbO_2$ with aid of Chart IX		
	Total CO_2 Content		Plasma NaCl	Total CO_2 Content		Plasma NaCl	Total CO_2 Content		Plasma NaCl
	Whole Blood	True Plasma		Whole Blood	True Plasma		Whole Blood	True Plasma	
m.m. Hg	vol. per cent	vol. per cent	mgs. per 100 cc.	vol. per cent	vol. per cent	mgs. per 100 cc.	vol. per cent	vol. per cent	mgs. per 100 cc.
20	30.4	42.2	518	31.2	42.9	515.7	31.8	43.4	514.4
40	38.3	48.7	507	39.2	49.6	504.7	39.9	50.2	502.8
70	48.1	59.7	505	49.6	61.0	502.2	51.7	61.8	500.6

A chart has now been constructed on which have been drawn the following lines: (1) the axis of ordinates representing volumes percentage of carbon dioxide contained in the whole blood or plasma, (2) the axis of abscissae representing carbon dioxide tensions, (3) the carbon dioxide solubility curve for the whole blood or plasma, (4) percentage acidity lines for whole blood or plasma. This is the skeleton chart which may be used for any blood and on which is to be constructed the series of lines peculiar to a specific case. These special lines are the carbon dioxide dissociation curves, the oxygen isobars and the chloride isopleths. On the appropriate dissociation curves the loci of arterial and venous points are to be located. The construction of these lines and the location of the arterial and venous points may be conveniently explained by following the steps actually taken in preparing the chart of a specific case.

THE CARBON DIOXIDE CURVES

In Table XI are recorded the necessary data (v. s.) obtained from the analysis of the blood from a case of pneumonia. As pointed out above, however, it is necessary to modulate the

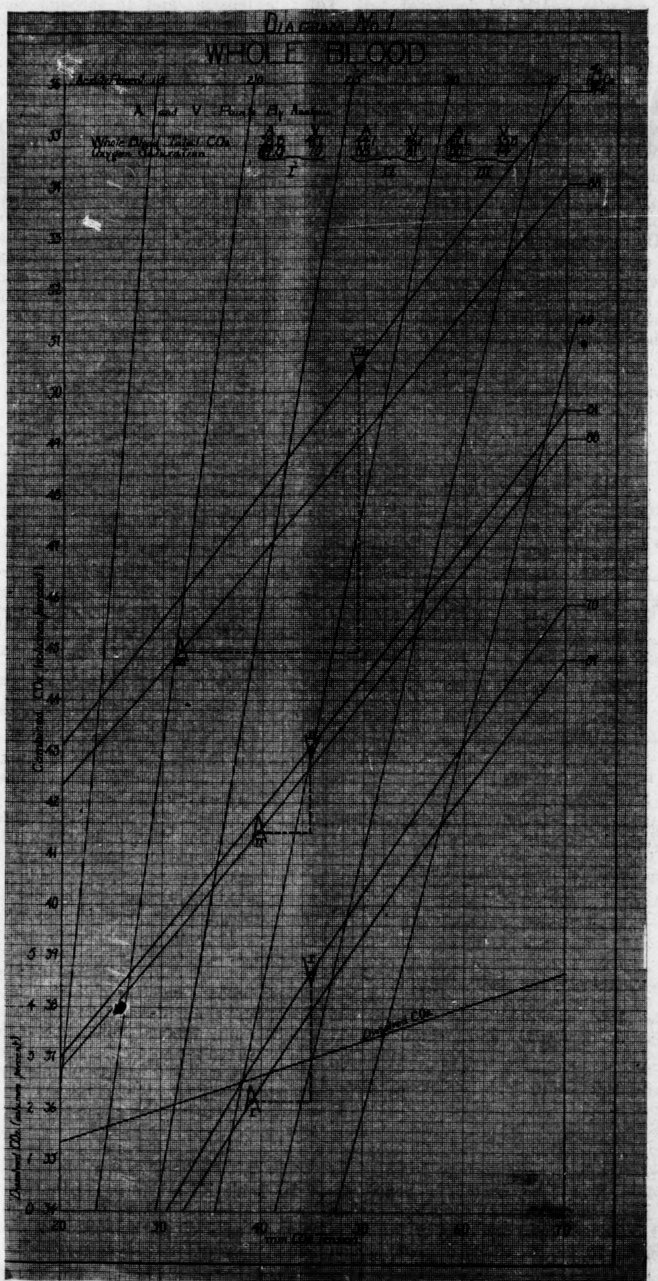


Diagram No. 1
WHOLE BLOOD
A and V Points By Analysis
Whole Blood Total CO2
Oxygen Saturation
I
II
III
Combined CO2 (volumes percent)
Dissolved CO2 (volumes percent)
Dissolved CO2
mm CO2 tension
20
30
40
50
60
70
34
35
36
37
38
39
40
41
0
1
2
3
4
5

actual values, obtained from analysis of blood exposed to carbon dioxide tensions of 20, 40 and 70 mm. and oxygen tensions of 155, 151 and 145 mm. respectively, to values which would have been obtained had the blood been exposed to the same tensions of carbon dioxide but to oxygen tensions which would have permitted the degree of oxygen saturation actually found by analysis of the arterial and venous blood specimens, in this case, 89% and 70% respectively. This was done by the aid of Chart IX. Thus the actual value found for the carbon dioxide content of the whole blood exposed to a tension of 20 mm. of carbon dioxide and 155 mm. of oxygen was 30.4 volumes per cent. But at an oxygen tension corresponding to 89% saturation at a carbon dioxide tension of 20 mm. it is seen from inspection of the chart (line D in panel labelled "20 mm.") that the whole blood would contain 2.66% more carbon dioxide. Now 2.66% of 30.4 are 0.8. This quantity added to 30.4 yields 31.2 which is the corrected value for the carbon dioxide content of blood which, after exposure to an atmosphere in which the carbon dioxide tension was 20 mm., has an oxygen saturation of 89%. In a similar manner, the other corrected values for the carbon dioxide contents recorded in Table XI were reckoned. From these values two carbon dioxide dissociation curves were plotted on the chart, the upper one corresponding to blood 89% saturated with oxygen, the lower one corresponding to blood 70% saturated with oxygen. The curves here referred to are the lowermost of the three series of curves represented on this diagram. (Diagram I, series I.) The upper series of curves were plotted from data obtained from the same case of pneumonia immediately after crisis (series II) and just before leaving the hospital (series III).

It must again be emphasized that the corrected values for carbon dioxide content enumerated in Table XI represent values for total carbon dioxide content. But it is desired that the vertical distance of any point in the carbon dioxide dissociation curve above the axis of abscissae shall represent combined carbon dioxide. It is therefore necessary in locating the three points on the 20, 40 and 70 mm. lines, through which the curve is to be drawn, that the vertical distance representing total carbon dioxide content shall be measured not from the axis of abscissae but from the temporary negative carbon dioxide solubility line.

After drawing the two carbon dioxide dissociation curves for the whole blood the arterial (A) point and the Venous (V) point were placed on these curves according to the following procedure. On a piece of stiff paper two points were marked such that the distance between them corresponded to 38.8 volumes per cent. of carbon dioxide, the total carbon dioxide content of the sample of arterial blood whose oxygen saturation was 89%. Maintaining, then, one of the points on the negative carbon dioxide solubility curve, the edge of the strip being kept parallel to the axis of ordinates, the strip was moved towards the 89% dissociation curve until the other point touched this curve. This point on the curve, the arterial point, was labelled "A." In a similar manner the venous point, "V," was located on the dissociation curve corresponding to 70% oxygen saturation.

After locating the arterial and venous points on their appropriate curves interrupted lines were drawn measuring the vertical and the horizontal differences between the levels of these two points.

By inspection of the diagram, now, the following inferences may be made: (1) the general shape of the carbon dioxide dissociation curves and their relative levels, (2) the percentage acidity of the arterial and of the venous whole blood, (3) the apparent carbon dioxide tensions to which the arterial and venous blood specimens were exposed and the difference between these tensions, (4) the difference in volumes per cent. between the combined carbon dioxide content of the arterial and the venous whole blood. In this particular case the values obtained from inspection of the diagram are as follows:

	Acidity Per Cent.	Apparent Tension Carbon Dioxide.	Combined Content Carbon Dioxide.
		mm.Hg.	Vols. P.C.
Venous Whole Blood:	28	45.0	48.8
Arterial Whole Blood:	24	36.5	45.8
Difference:	4	8.5	3.0

In a precisely similar manner the carbon dioxide dissociation curves for plasma separated from whole blood exposed to 20, 40 and 70 mm. of carbon dioxide tension were drawn and on these curves were located the arterial and venous points corresponding respectively to plasma separated from the arterial blood and plasma separated from the venous blood. (Diagram I, Plasma.)

For many purposes no further elaboration of the diagram is desirable, but it is possible by very little additional labor to study the changes in the oxygen tension and the shift in the chloride ion content of the plasma as the blood

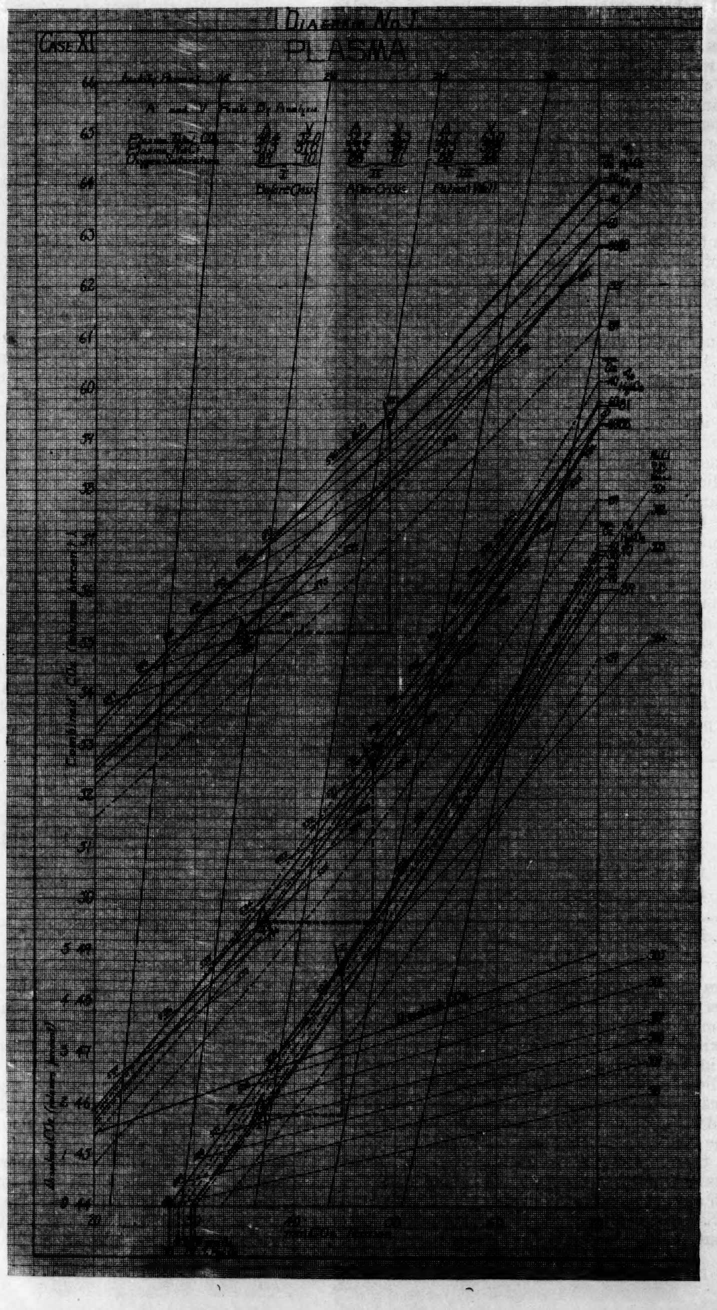


Diagram No. 1
Case XI
PLASMA
A and V
I
Before Crisis
II
After Crisis
III
Patient Well
66
65
64
63
62
61
60
59
58
57
56
55
54
53
52
51
50
5 49
4 48
3 47
2 46
1 45
0 44

passes from arterial to venous conditions. The graphs for oxygen tension and for sodium chloride content of the plasma may, of course, be plotted either on the diagram of the plasma or on that of the whole blood. The following description refers to plasma.

OXYGEN ISOBARS

Again it will be convenient to explain the manner of constructing these lines by describing what was done in the particular case above referred to. The construction of the oxygen isobaric lines will be considered first. Inasmuch as the effect of diminishing the oxygen tension from 155 mm. (or 151 or 145 mm., as the case may be) to any given tension on the carbon dioxide content of the whole blood or plasma kept at a constant carbon dioxide tension has been determined, it is possible to calculate for any oxygen tension the quantity of carbon dioxide at 20, 40 and 70 mm. of carbon dioxide tension which the blood would contain were it equilibrated in an atmosphere in which the oxygen tension was the one selected instead of one in which the oxygen tension was 155, 151 or 145 mm. Three points are thus located and the curve drawn through these is the required oxygen isobar. It is not necessary, as a rule, to draw oxygen isobars for differences of tension smaller than 10 mm. nor, of course, is it necessary to draw lines representing oxygen tensions which, under the circumstances, would probably not be encountered.

TABLE X

Percentage Increase of CO_2 Content of Plasma Compared with Decreasing Oxygen Tension at Constant CO_2 Tensions of 20, 40 and 70 m.m. Hg.

(Taken from Chart IX)

Oxygen Tension	20 m.m. CO_2 Tension	40 m.m. CO_2 Tension		70 m.m. CO_2 Tension
	Increase in CO_2 Content	Increase in CO_2 Content	$HgbO_2$	Increase in CO_2 Content
m.m. Hg	percent	percent	percent	percent
90	1.07	1.19	92.6	2.05
80	1.24	1.46	91.2	2.32
70	1.40	1.75	89.3	2.62
60	1.60	2.08	86.6	2.98
50	1.78	2.50	82.2	3.34
40	2.02	2.96	75.5	3.68
30	2.40	3.47	65.2	4.00
20	3.03	4.02	47.0	4.30
10	3.58	4.44	20.0	4.54

Now since the relative levels of the two carbon dioxide dissociation curves are determined by the percentage saturation with oxygen of the arterial and venous blood specimens and since the arterial and venous points must fall on these curves it follows that the oxygen isobars must be selected so that these points will fall within the space between the highest and the lowest oxygen tensions selected.

Moreover, inasmuch as the mean carbon dioxide tension between arterial and venous blood is, as a rule, near the 40 mm. line, it is evident that those oxygen isobars which, on the 40 mm. line, pass through points representing percentage oxygen saturations which include those of the arterial and the venous blood will include also the appropriate oxygen isobars for arterial and venous blood. For convenience a table (Table X) has been constructed based on the data of Chart IX and showing the oxygen isobars corresponding to different oxygen percentage saturation values at 40 mm. of carbon dioxide tension. In the particular case under consideration the percentage saturation of the venous blood was 70 and of the arterial blood 89. From Table X, then, it is obvious that oxygen isobars for 70, 60, 50, 40 and possibly also for 30 mm. tension should be constructed. As an example, the procedure adopted in locating the 70 mm. oxygen tension line may be followed.

It is evident from inspection of Table X that if the oxygen tension to which blood is exposed be reduced from 155 mm. to 70 mm., at a carbon dioxide tension of 40 mm., the percentage increase in the carbon dioxide content of the plasma will be 1.40. The carbon dioxide content of the plasma separated from blood equilibrated at a carbon dioxide tension of 20 mm. and an oxygen tension of 155 mm. was 42.2 volumes per cent. (Table XI.) Now 1.4% of 42.2 are 0.6 and this value added to 42.2 gives 42.8, the total carbon dioxide content of the plasma at a carbon dioxide tension of 20 mm. corrected to an oxygen tension of 70 mm. At 40 mm. and at 70 mm. the corrected values are similarly found to be 49.5 and 61.3 respectively. Accordingly, on the 20, 40 and 70 mm. vertical carbon dioxide tension lines points were marked at distances from the negative carbon dioxide solubility line corresponding respectively to 42.8, 49.5 and 61.3 volumes percent. of carbon dioxide. Through these three points a curve was drawn every point in which represents an oxygen tension of 70 mm., the 70 mm. oxygen isobar. In a similar manner the oxygen isobars for 60, 50, 40 and 30 mm. were constructed.

THE ISOPLETHS FOR SODIUM CHLORIDE

Owing to the fact that the absolute quantities of sodium chloride in the plasma vary in every case it is necessary to calculate a different set of values for every diagram constructed if comparisons with values obtained from analyses of arterial and venous plasma specimens are to be made. Before describing the method of constructing the sodium chloride isopleths it will be desirable briefly to comment on the theoretical significance of these lines.

The earlier work on the relation of the shift in chloride ions from plasma to corpuscles with increasing carbon dioxide tension led to the view that for a given increment in the bicarbonate content of the plasma a definite shift in chloride ions from plasma to cells took place. That is to say, for every volume percent. of bound bicarbonate gained by the plasma a definite number of milligrams of chloride ions entered the cells. If this were the case, however, it would necessarily follow that the chloride isopleths would be strictly parallel to the bicarbonate isopleths. From theoretical considerations L. J. Henderson has shown not only that the chloride isopleths are not parallel to the bicarbonate isopleths but also that the former are not straight lines. Experimental evidence, too, confirms this and our own data are such as to yield values for chloride lines that correspond to those presented in the diagrams of Henderson, Bock, Field and Stoddard[15] though our method of constructing these lines was entirely different.

As previously explained and illustrated in Chart IV we have, from numerous analyses, determined the relationship between the percentage increase in the plasma bicarbonate and the percentage decrease in the plasma chloride content, a relationship such that an increase of about 4% in plasma bicarbonate content cor-

Data Obtained From Analyses Of Specimens Of Arterial And Venous Blood And Data Obtained From Inspection Of The Blood Diagram (The Latter Are Found In Columns Marked With An Asterisk. Blood Specimens Were Taken Under Oil.

Table XI-B.

		Oxygen Capacity volumes percent	Oxygen Content volumes percent	Oxygen Saturation. percent	Volume of Red Cells. percent	* Whole Blood CO_2 Content at 40 mm. CO_2 Tension and Arterial Saturation	* Plasma CO_2 Content at 40 m.m. CO_2 Tension and Arterial Saturation	Difference	NaCl mgs. per 100 c.c.	* NaCl Isopleth Difference (Plasma)	CO_2 Content. volumes percent	Whole Blood Difference in CO_2 Content	Plasma Difference in CO_2 Content	Whole Blood Difference in CO_2 Content less Plasma CO_2 Difference	* Percent Acidity	* Apparent Oxygen Tension	* Apparent CO_2 Tension
Arterial	Whole Blood	20.2	18.0	89	38	38.3			429		38.8				31.2	65.0	39.1
	Plasma						48.7		515		48.4				24.2		
	Difference								86		9.6				7.0		
Venous	Whole Blood	20.2	14.2	70	38				429		41.7				33.4	35.0	44.9
	Plasma								510		52.0				28.0		
	Difference								81		10.3				5.4		
Difference between Arterial and Venous Whole Blood or Plasma			3.8	19	0			10.4		4		2.9	3.6	-0.7		30.0	5.8

responds approximately to a decrease of about 1% in the plasma chloride content. It is not here claimed either that our data are finally accurate or that even this method of comparing bicarbonate changes with chloride ion shifts will eventually turn out to be an entirely rational procedure. All that can be said is that in the present status of our knowledge, the data and the empirical method of representing them are in accord with the data of other observers and with the behavior of the chlorides represented in the diagrams of Henderson, Bock, Field and Stoddard.

Certain theoretical possibilities, however, should be borne in mind. If the percentage decrease in the sodium chlorides content of the plasma is proportional to the percentage increase in the bicarbonate content of the plasma then two conclusions are necessary: first, that at a fixed oxygen percentage saturation of the whole blood and with a given increase in the oxygen tension, of two blood specimens of unequal initial bicarbonate content, the one with the lesser will show, in absolute amount, the greater decrease in the chloride content of the plasma for each volume percent. of bicarbonate gained by the plasma; and, second, that at a constant carbon dioxide tension, an increase in the oxygen percentage saturation of the blood will permit a decrease in the bicarbonate content of the plasma and chloride ions will pass from corpuscles into plasma. These conclusions are, of course, borne out by experimental evidence and possibly the following explanation may account for them.

It has been suggested by Buckman and Edwards[16] that amino groups in proteins act somewhat as ammonia does in the Solvay process for the manufacture of sodium bicarbonate from carbon dioxide and sodium chloride and that the reaction between protein, carbonic acid and sodium chloride may be represented thus:

$$RNH_2 + NaCl + H_2CO_3 \leftrightarrows RNH_2HCl + NaHCO_3$$

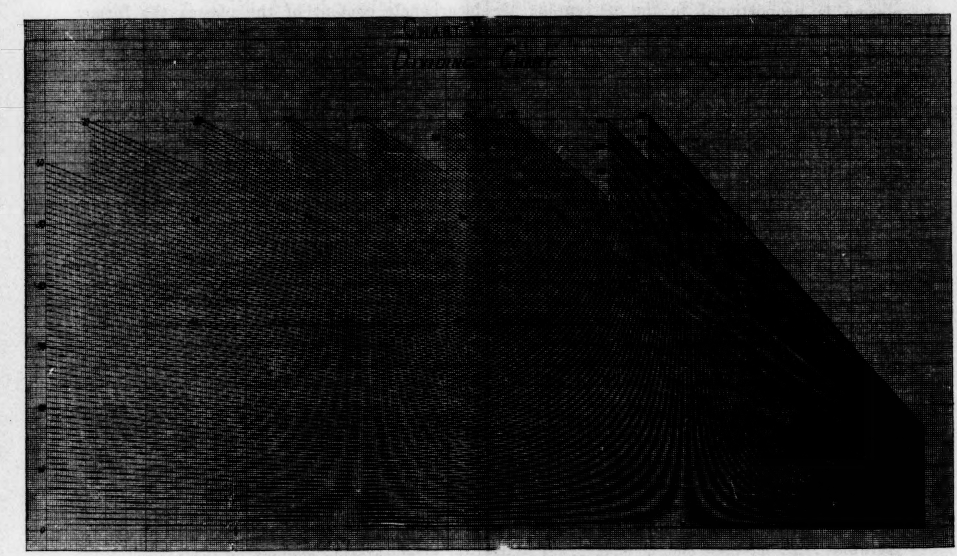
Now any influence operating to increase the hydrogen ion concentration of the medium in which this reaction takes place will obviously tend to suppress the dissociation of the amino group and hence tend to inhibit the formation of sodium bicarbonate.

This reaction, which is presumably a general one, may be assumed to take place in the plasma and between red cells and plasma. In the former case analysis of the plasma exposed to varying tensions of carbon dioxide show no change in chloride ion content because from the point of view of chloride analysis amino hydrochloride and sodium chloride are indistinguishable. In the latter case, however, a diminution of plasma chloride is noticed as the concentration of carbonic acid increases because hydrochloric acid has become bound to cell protein. If, now, two specimens of blood of unequal plasma bicarbonate content be exposed to the same increase in carbon dioxide tension and to such an increase in oxygen tension that no change in the oxygen percentage saturation of either occurs, then the hydrogen ion concentration within the cells will tend to remain constant but the plasma of the specimen with the lesser initial plasma bicarbonate content will suffer the greater increase in hydrogen ion concentration because in this specimen the ratio of dissolved to combined carbon dioxide will be the greater. This means that in the specimen with the lesser initial plasma bicarbonate content conditions are more favorable for reaction between plasma and cells than in the specimen with the greater initial bicarbonate content and the result will be that for a given increment in bicarbonate content of the plasma the former specimen will show the greater diminution of plasma chloride content with increasing carbon dioxide tension.

On the other hand, at a constant carbon dioxide tension, oxygenation of the blood permits an increase in the hydrogen ion concentration of the cells and this in turn tends to inhibit the reaction between cells and plasma so that there occurs not only a diminution of plasma bicarbonate but also a gain in plasma content of chloride ion proportional to the increase in percentage saturation of the blood. This, in turn, is proportional to the percentage decrease of plasma bicarbonate. In either case, whether or not the method of representing by percentages the relation between chloride shift and plasma bicarbonate changes, which at present seems convenient and sufficiently accurate, will turn out to be mathematically rational, does not matter. The important consideration is that this relation should not be represented by a simple ratio between absolute quantities of chloride and bicarbonate ions but by an expression in which the ratio of the increment of the bicarbonate content of the plasma to the initial total bicarbonate content is equated to the ratio of the increment in the chloride shift to the initial total chloride content of the plasma.

From what has been said it follows, also, that if the oxygen tension be increased so that with increasing carbon dioxide tension the bicarbonate content of the plasma be kept constant, then, as the percentage oxygen saturation of the blood increases, the chloride ion content of the plasma will increase even though the plasma bicarbonate content remain unchanged. The gain in plasma bicarbonate is offset by reversal of the reaction between plasma and cells.

In view of these considerations the chloride isopleths assume a new importance. As pointed out by L. J. Henderson, they limit the conditions under which a shift of electrolytes between plasma and cells can take place. But, in a certain sense, the chloride isopleths also limit the conditions under which the transfusion of water between plasma and cells can occur. This will be evident from a consideration of the above equation for the reaction between protein, sodium chloride and carbonic acid. If conditions are such as to permit this reaction to take place between cells and plasma, then, since sodium bicarbonate displaces sodium chloride, molecule for molecule, in the plasma and since these two salts are of the same order of degree of dissociation, the net change in the concentration of particles in the plasma will be very slight. On the other hand, as chloride ions enter the cells an increase in concentration of particles within the cells will occur. As a result water will pass from plasma into cells. In general, the quantity of water that passes from plasma into corpuscles with increasing carbon dioxide tension is thus, in a measure, proportional to the magnitude of the change in concentration of chloride ion in the plasma.

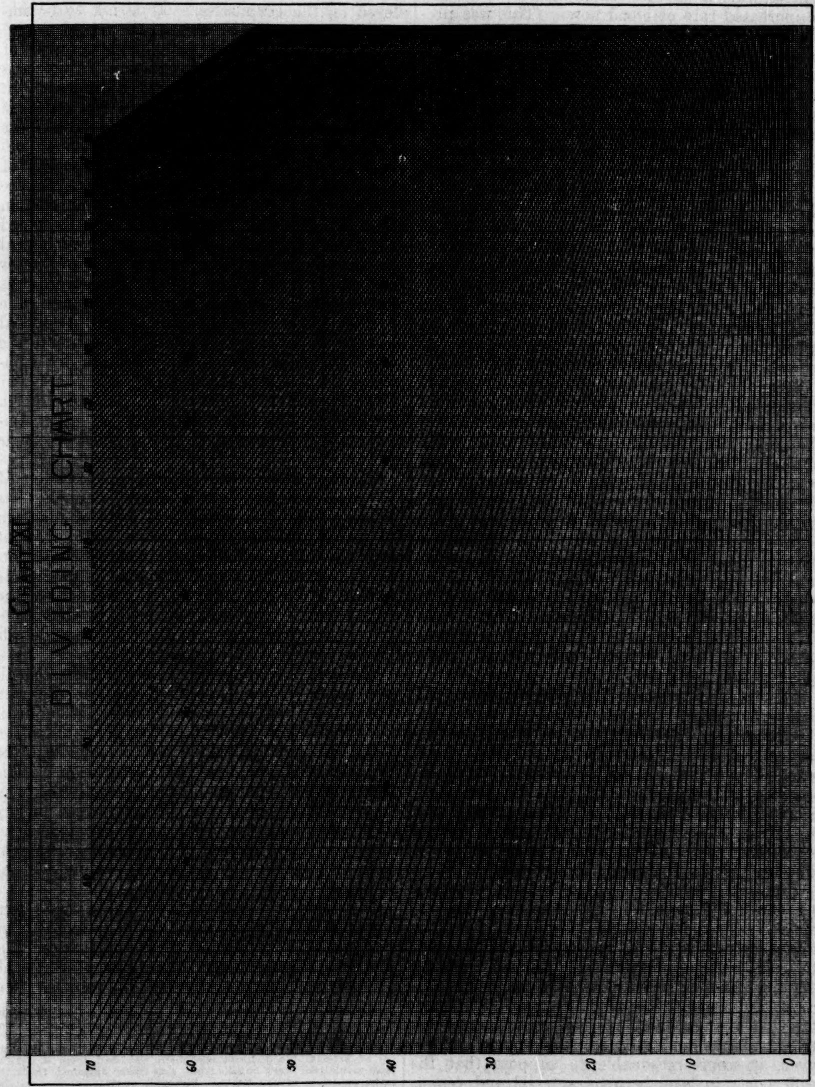


In describing the method of constructing the chloride isopleths it will once again be convenient to review the procedure followed in drawing the lines of the diagram already considered.

The data presented in Table XI show that the chloride ion contents, reckoned as sodium chloride, of specimens of plasma separated from blood equilibrated at tensions of carbon dioxide of 20, 40 and 70 mm. Hg were 518, 507 and 505 mgm. of sodium chloride per 100 c.c. of plasma, respectively. These values when corrected according to Chart IX become, for 89% oxygen saturation, 515.7, 504.7 and 502.2, and, for 70% oxygen saturation, 514.4, 502.8 and 500.6 mgm. per 100 c.c. of plasma.

At the intersection of the 20, 40 and 70 mm. tension lines the carbon dioxide dissociation curve corresponding to 89% oxygen saturation points were marked representing sodium chloride concentrations of 515.7, 504.7 and 502.2 mgm. per 100 c.c. of plasma respectively. In the same way, at the intersections of the 20, 40 and 70 mm. tension lines with the carbon dioxide dissociation curve corresponding to 70% oxygen saturation points were marked representing sodium chloride concentrations of 514.4, 502.8 and 500.6 mgm. per 100 c.c. of plasma respectively. By means of the dividing charts (Charts X and XI) that portion of the carbon dioxide dissociation curve corresponding to 89% oxygen saturation which lies between the points marked 515.7 and 504.7 was divided into 110 parts and points representing 515, 514, 513 etc.—505 mgm. of sodium chloride were marked. Similarly, that portion of the curve lying between the points marked 504.7 and 502.2 was divided into 25 parts and points representing 504, 503, 502 mgm. of sodium chloride were marked. In the same way, points were located and marked on the carbon dioxide dissociation curve corresponding to 70% oxygen saturation. Points on the two curves having the same values were connected, then, by straight lines, the sodium chloride isopleths. Although it is probable that these isopleths are not absolutely straight lines the error involved in disregarding their curvature is inconsiderable.

With the construction of the sodium chloride lines the diagram for the plasma is completed. The diagram for whole blood is constructed according to the same principles.



INTERPRETATION OF THE DIAGRAM

Having thus constructed alignment diagrams for the whole blood and plasma of a particular individual, what inferences may be drawn? In the first place, it is possible to determine the apparent tensions of oxygen and carbon dioxide to which the blood was exposed; apparent because it is assumed that the blood flow was sufficiently slow to permit complete exchange of gases. As a matter of fact, as Bock[17] has shown, changes in the rate of flow of the blood will be reflected in the relation between the arterial and venous percentage saturations of the blood with oxygen, abnormally high oxygen saturation of the venous blood indicating an increased rate of blood flow. This was presumably the case in the patient here studied not only at the height of the disease from which he suffered, pneumonia, when the percentage oxygen saturations of arterial and venous blood specimens were 89 and 70 ,respectively, but even twenty-four hours after crisis had occurred (Diagram I, middle series of curves) when the percentage saturation values became 81 for the venous blood and 88 for the arterial blood. In the present instance the apparent oxygen tension to which the blood was exposed in the lungs was 65 mm. and in the tissues 35 mm. Hg. The apparent carbon dioxide tension in the lungs was 39.1 mm. and in the tissues, 44.9 mm. Hg. The low venous carbon dioxide tension and the small difference between the two carbon dioxide tensions are due probably to an increased rate of blood flow.

Secondly, the level and shape of the carbon dioxide dissociation curves are apparent from inspection. The differences in level between these curves at the three periods of observation of the patient are plainly exhibited in the diagram. In this illustration no noteworthy differences in shape are evident but it is well known that such differences do occur and it has been suggested that the shape of the carbon dioxide dissociation curve may have an important bearing in determining the limits of an efficient circulation. Attention should also be directed to the vertical interrupted lines indicating the differences between the bicarbonate contents of both arterial and venous whole blood and true plasma.

Thirdly, the hydrogen ion concentrations, pH values or acidity percent. values—it makes little difference what numbers or labels are attached to the lines—of both arterial and venous whole blood and plasma are obvious from inspection of the chart. The actual values here shown illustrate the fallacy, often pointed out by others, of assuming that the carbon dioxide combining power of the plasma is necessarily a measure of its hydrogen ion concentration.

Finally, it is of the greatest interest to observe the differences in level between the arterial and venous chloride isopleths. In the three observations here reported the differences in level were approximately 3.5, 4.5 and 6.0 mgm. As mentioned above the isopleths indicate conditions under which no passage of electrolytes and probably no passage of water between plasma and cells can take place. Moreover, it seems reasonable to suppose that the differences in level between arterial and venous chloride isopleths are, in some way, representative of the magnitudes of the possible shifts of electrolytes and water between plasma and corpuscles. A small difference in level would suggest that the burden of carrying electrolytes, including sodium bicarbonate, is placed on the plasma whereas a great difference would indicate that a relatively greater burden is placed on the corpuscles. It is not to be imagined, of course, that the actual values, found by analysis, for the sodium chloride contents of arterial and venous true plasma will correspond to those indicated by the difference in levels indicated by the isopleths. Not only are the differences too small to be outside the limits of experimental error but there is also an obvious theoretical reason why the two sets of values should not agree: the isopleth values are determined by analysis of blood manipulated in vitro whereas the arterial and venous blood specimens have been subjected to physiological compensations, including absorption of water from the tissues, in the body. Moreover, it is obviously futile, in view of the small magnitude of these differences and the present technical obstacles to accuracy, to attempt to estimate the extent of physiological compensation by comparing the two sets of values.

The quantitative information derived from a study of the alignment diagrams has been summarized and presented in tabular form in Table XI.*

Although the nomographic representation of the arterio-venous cycle above described is probably the most convenient form of diagram for many purposes, it has occurred to us that some advantage would be gained by constructing a diagram in which changes in the whole blood and plasma could be represented on the same chart. For this purpose we have described another type of diagram, the details for the construction of which follow.

Skeleton for diagram. Millimeter coördinate paper of the type used for the diagram above described is recommended. The ordinates represent percentage oxygen saturation of the hemoglobin, allowing 5 mm. as the equivalent of one percent. saturation. The degree of oxygenation of the arterial and venous blood must, of course, be determined in any given case and the chart must be made long enough in the direction of the ordinates so that these two percentage saturation values will be included.

The abscissae represent carbon dioxide tension in millimeters of mercury, allowing 10 mm. of distance to represent 1 mm. of tension. The abscissae represent also quantities of physically dissolved carbon dioxide in volumes percent., values for which may be obtained from Table XII in which are shown the volumes per cent. of dissolved carbon dioxide in the whole blood and in the plasma corresponding to definite tensions of carbon dioxide.

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*A very general criticism must here be made regarding the interpretation of all values obtained by the analysis of blood from a peripheral vein. Whereas blood specimens obtained from an artery such as the radial may be looked upon as typical of arterial blood, blood specimens obtained from a peripheral vein differ very much from specimens obtained from the right ventricle of the heart. Although successive observations on blood obtained from the peripheral vein of a given individual may yield comparable data, the evaluation of these data must be looked upon as empirical.

It goes without saying that in any study of the blood from the point of view of its gaseous content every precaution must be taken to prevent disturbances in the pulmonary ventilation during the taking of the specimens.

The oxygen isobaric lines may be easily constructed by drawing curves through points on the 20, 40 and 70 mm. carbon dioxide tension lines that represent the same oxygen tension at different oxygen percentage saturations. Data for the construction of these isobars are given in Table XIII, compiled from Chart IX.

In any given case there remain to be traced for whole blood and plasma the bicarbonate isopleths and the isohydric lines and for the plasma alone the chloride isopleths.

In order to construct the bicarbonate isopleths it is first necessary to mark at the intersections of the vertical 20, 40 and 70 mm. carbon dioxide tension lines with the horizontal lines representing the percentage oxygen saturations of the arterial and venous blood specimens the two sets of values for total carbon dioxide content of the blood corresponding to the arterial and venous percentage oxygen saturations at 20, 40 and 70 mm. of carbon dioxide tension. In the present instance, in constructing the plasma bicarbonate isopleths, the points of intersection of the 89% oxygen saturation line with the 20, 40 and 70 mm. carbon dioxide tension lines have values, respectively, of 42.9, 49.6 and 61.0 volumes per cent. of total carbon dioxide content, these being the total carbon dioxide content values for the plasma corrected to 89% oxygen saturation. (See Table XI.) Similarly, on the 70% oxygen percentage saturation line, the corresponding points of intersection with the carbon dioxide tension lines represent values of 43.4, 50.2 and 61.8 volumes percent. of total carbon dioxide. By interpolation, the values on the horizontal lines points of equal value on the line representing 89% oxygen saturation and on the line representing 70% oxygen saturation may be located and connected by straight lines. These straight lines do not represent the true bicarbonate isopleths for the latter are slightly curved lines even in physiologically significant parts of the diagram and are markedly curved in regions of high oxygen tension. It is advisable, therefore, unless the difference between the percentage saturation of arterial and venous blood is slight, to locate, by means of Chart IX and then by interpolation as above described, points on one or more additional horizontal lines and then to connect the points of equal value by a smooth curve. These bicarbonate isopleths represent values for total carbon dioxide content. Values for combined carbon dioxide content must be ascertained by consulting the scale at the top of the diagram.

The points of intersection of the oxygen percentage saturation lines with the carbon dioxide tension lines represent values not only for total carbon dioxide content as explained above but also values for hydrogen ion concentration or percentage acidity. Since the carbon dioxide tension for each of these points is known and since the quantity of dissolved carbon dioxide is proportional to the carbon dioxide tension it

TABLE XII

Carbon Dioxide Tensions and Corresponding Values for Dissolved Carbon Dioxide in the Whole Blood and Plasma

Dissolved CO_2	Carbon Dioxide Tension	
	Plasma	Whole Blood
vol. percent	m.m.Hg	m.m.Hg
1.0	14.04	14.88
2.0	28.09	29.76
3.0	42.14	44.64
4.0	56.18	59.52
5.0	70.23	74.41
6.0		89.29

follows that the bound carbon dioxide or bicarbonate ion content is also determined. Then by use of equation VII or VIII (page 1007), the percentage acidity value for any given value of carbon dioxide content at a given carbon dioxide tension can easily be calculated. By means of Chart XII in which the subtraction of dissolved from total carbon dioxide is automatically accomplished it is a simple matter to calculate isohydric points for a series of horizontal lines, as in the case of the bicarbonate isopleths, and by drawing smooth curves through these points to construct the percentage acidity lines.*

TABLE XIII

PERCENTAGE OXYGEN SATURATION OF HEMOGLOBIN AT VARYING OXYGEN TENSIONS AND AT CARBON DIOXIDE TENSIONS OF 20, 40 AND 70 M.M. HG.

(Taken from Chart I)

Oxygen Tension m.m. Hg	Percentage Oxygenation		
	20 m.m. CO_2	40 m.m. CO_2	70 m.m. CO_2
155	96.6		
151		95.7	
145			94.3
100	95.6	93.6	91.4
90	94.8	92.6	90.0
80	93.8	91.2	88.2
70	92.5	89.2	85.4
60	90.8	86.6	80.4
50	88.6	82.2	73.8
40	85.3	75.5	65.4
30	78.5	65.2	54.6
20	60.0	47.0	41.0

The sodium chloride isopleths are constructed in a similar manner. Three series of values for the sodium chloride content of the plasma at 20, 40 and 70 mm. of carbon dioxide tension are obtained by correcting, through Chart IX, the values found on analysis to those corresponding to various percentage saturations of the whole blood with oxygen. Then by interpolation a series of values is located on each of the several horizontal lines chosen and by connecting points of equal value by smooth curves the sodium chloride isopleths are drawn. The abrupt change in the direction of these curves at about the 40 mm. carbon dioxide tension line is due to the flattening out of the carbon dioxide dissociation curve which occurs at about this point and which is shown in the type of diagram first described. Again, here as in the other type of diagram, the declination of the sodium chloride isopleths from the bicarbonate isopleths is illustrated and is due to the same causes discussed above.

Since bicarbonate isopleths for both whole blood and plasma are here represented on the same chart the loci of the arterial and venous points may be determined if only the percentage oxygen saturation values and the contents of total carbon dioxide of both arterial and venous blood specimens are known. Theoretically, the same considerations would hold for the type of diagram first described but in that case greater distances between all the curves would have to be allowed. Thus the first type of diagram possesses the advantages of simplicity of construction and of retaining the shape of the carbon dioxide dissociation curve of the blood. Its disadvantage is the difficulty of representing changes in both the whole blood and the plasma on the same chart. The diagram of the second type is more economical of space and permits the location of the arterial and venous points without recourse to analysis of the plasma of arterial and venous blood specimens for carbon dioxide. On the other hand, the shape of the dissociation curve is not shown in the manner in which it is customarily represented. Moreover, more draughtsmanship is required in the second type of chart than in the first, though this objection might be overcome by having printed a series of skeleton charts which could be used for any case.

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*In using Chart XII it should be noted that line A-B refers to plasma and line C-D refers to whole blood.

SUMMARY

1. There are presented in this paper oxygen dissociation and carbon dioxide dissociation curves of oxalated human blood drawn from original data, and also curves showing the relative shift in the sodium chloride content of the plasma with increasing bicarbonate content of the blood.

2. From these data a nomogram has been constructed, and is here presented, showing quantitatively the association of changes in the carbon dioxide tension and oxygen percentage saturation with oxygen tension, bicarbonate content of whole blood and plasma, hydrogen ion concentration and sodium chloride content of plasma.

3. Two methods, based on the relationships shown in this nomogram, have been described for the construction of diagrams in which are aligned the following factors:—carbon dioxide tension and bicarbonate content of whole blood and plasma, oxygen tension and oxygen percentage saturation of the whole blood, sodium chloride content of the plasma, and hydrogen ion concentration of the whole blood and plasma. The method of determining the loci of arterial and venous points on these diagrams has been described and illustrations of the use of such charts as computing diagrams have been given.

4. Finally, the significance of each of these factors in the physico-chemical system of the blood has been discussed and the importance which might attach to this method of analysis in the study of disease has been intimated.

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A TUBERCULOSIS SURVEY

Dr. Murray P. Horwood, Assistant Professor of Biology and Public Health at the Massachusetts Institute of Technology, has just completed his Tuberculosis Survey for the Boston Tuberculosis Association. Dr. Horwood is the author of a book on Public Health Surveys, and has conducted surveys in Philadelphia and other cities. Recently, he has studied Health Conditions in New England cities, for the American Child Health Association.

Among the activities studied are the work of the official and voluntary agencies in the city. These include the Massachusetts State Department of Public Health, the State Department of Public Welfare, the Boston Health Department, the Boston School Department, the Boston Sanatorium, the Boston Tuberculosis Association, and other agencies interested in the problem of tuberculosis.

Dr. Horwood has made a thorough analysis of vital statistics on tuberculosis, the adequacy of the dispensary, sanatorium and hospital facilities for the instruction and care of patients with tuberculosis, the adequacy and facilities of the case-finding machinery, open air classes, nutrition work, health education, together with milk supply, and the examination of food handlers.

This work was conducted with the coöperation of the above mentioned agencies.

Dr. Horwood's report will soon be ready for publication, and from the large number and variety of the recommendations which have been submitted, we may be assured that the tuberculosis program in Boston will be conducted along highly scientific and efficient lines for many years to come.

This report will be placed in the hands of a responsible committee which will consider ways and means of introducing the recommendations.

DEATHS DUE TO SYPHILIS IN FRANCE

Twenty thousand infant deaths, 40,000 abortions and 80,000 deaths among adults is the toll exacted by syphilis in France each year, according to the Minister of Labor, Hygiene and Social Welfare as reported in a recent number of *The Lancet* (London). The government is undertaking a campaign of public enlightenment and is instituting preventive measures for which 4,000,000 francs are appropriated annually. The Minister states that progress is taking place and points to the fact that syphilis is no longer regarded as a disease to be hushed up.—*United States Public Health Service.*

Case Records of the Massachusetts General Hospital

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICO-PATHOLOGICAL EXERCISES

EDITED BY
RICHARD C. CABOT, M.D., AND HUGH CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 11481

MEDICAL DEPARTMENT

An American railway engineer sixty-nine years old entered August 7 complaining of shortness of breath of six years' duration. His father died of cancer. One brother dropped dead. His wife had had one miscarriage. Since childhood he had had frequent nosebleeds, less frequent recently. He had always been troubled with gas. Until he was sixty he drank two glasses of whiskey and three of beer a day, sometimes more, and smoked very heavily. That year he had an attack of "inflammatory rheumatism" lasting fifteen weeks. At fifty-seven and sixty-three he had pneumonia. For several years he had had some deafness. He drove his engine at high speed, under considerable nervous strain. He urinated five or six times by day and two or three at night. He had slight burning and occasional difficulty in starting the stream.

For thirty-seven years, ever since he had been an engineer, he had had dyspnea. For many years he had been obese. The dyspnea had been slight until his attack of bronchopneumonia six years before admission. Since that time he had cough with thick greenish sputum. Three years before admission he raised a little blood streaked sputum. The winter before admission his cough was so bad that he lost forty-eight pounds. He coughed chiefly at night, especially on lying down. Two years before admission he had another attack of pneumonia. Since that time the dyspnea had been worse. It was relieved by sitting up but aggravated by slight exertion and by lying down. Since February his appetite had been poor. For the past two weeks his feet had been swollen for the first time. For a month or more he had taken a tonic which had a little digitalis in it. Later conversation with his physician and his son showed that he had had one dram of digitalis infusion for four doses daily for five days and tincture of digitalis in uncertain quantity for two days, all consecutive and immediately preceding his admission. He stopped it the day before admission because he was much nauseated. His son insisted that the onset of the present illness was without pain.

Physical examination showed a very obese old man sitting propped up, with marked cyanosis and profuse sweating. The tongue showed a brown coat. The apex impulse of the heart was not found. The left border of dullness was 9.5 cm. from midsternum, 1.5 cm. outside the midclavicular line. The right border of dullness was 3 cm. to the right. The supracardiac dullness was 7 cm. The action was irregular and slow, with possible extrasystoles appearing very late in diastole or possible auricular fibrillation. There was a soft systolic murmur. The beats came through at different levels in taking the blood pressure, which was 140-145/90-95, but true alternation was not present. Electrocardiogram showed auricular fibrillation, rate 80, bigeminal pulse due to ectopic ventricular contractions, marked regularity of the ventricle. The lungs showed a few râles at both bases behind. The abdomen was very adipose, with bulging and dullness in the flanks. There was edema of the feet and legs up to the knees.

The temperature was 97.1° to 104°, the pulse 70 to 154, the respiration 14 to 38. The urine was cloudy, the specific gravity 1.018, a trace of albumin, 3 to 5 red blood corpuscles per high power field, amount 30 ounces on the one occasion recorded. The blood showed 10,000 to 19,950 leucocytes, 75 per cent. polynuclears, hemoglobin 80 per cent., 6,300,000 reds, platelets normal. No Wassermann is recorded.

The patient was given digifolin gr. iii every three hours for four doses. It produced vomiting and was discontinued. August 10 the patient died.

Note by the senior house officer, Dr. Norman Johnson. The patient had been removed from his engine because of a sudden attack of extreme dyspnea and collapse. There was no history of severe pain. He was driven by automobile sixty miles to his home, and a week later entered the hospital. Vomiting, though not marked, had been present daily during the week, worse during the two or three days immediately preceding admission. There was considerable question upon admission of over-digitalization until it was found that the infusion and not the tincture had been given during the past week. Hence digitalis was given very cautiously at first and was stopped on recurrence of nausea. Later data proved that too great caution was used in this respect.

DISCUSSION

BY DR. RICHARD C. CABOT

NOTES ON THE HISTORY

Summing up prior to the physical examination we have what sound on the whole like heart symptoms in an old man. Certainly at the end during these few days when he has been taking so much digitalis somebody thought he had heart trouble. But there is a good deal also to call attention to the lungs. He has had these long attacks of cough with purulent sputum, and as they follow an attack of pneumonia one wonders whether he did not get a bronchiectasis following that. At the same time it is perfectly possible that it is all heart and that passive congestion accounts for the cough. It is with those two things especially in mind that I shall begin looking for light in the physical examination.

NOTES ON THE PHYSICAL EXAMINATION

The heart shows no enlargement by percussion.

We get alternation almost always if we are on the watch for it in the process of taking a blood pressure. That saves our doing a pulse tracing and puts us in possession of a very important piece of prognostic information, because alternation is the worst form of disturbance of rhythm that we have.

The temperature was normal until the last day practically. During two-thirds of the time he was here it was an afebrile case.

There is no history of severe pain, that is nothing to suggest infarction of the heart.

DIFFERENTIAL DIAGNOSIS

Our guesses about his lungs, about there being organic disease there, possibly bronchiectasis, do not seem to be supported by anything that comes out in the physical examination. We do not hear of purulent sputum or of anything in the lungs that would not be accounted for by passive congestion. I guess we can say, therefore, that this is a heart case and not a lung case, and outside of these two organs there has been nothing to attract our attention.

There is no evidence of valvular disease or of syphilis. There is very little evidence of hypertension. I do not think one can say there was anything definite about the blood pressure as we get it here, but of course we are seeing him only at the end, and one often suspects that there has been hypertension in such cases before one sees the patient. I do not know anything better to suppose than that this was of the hypertensive type of heart trouble, the heart giving way from trying to keep up against that pressure.

There is something in the history to suggest that something rather sudden happened to his heart. There was a collapse, and of course he is at the age when we can get infarction with or without chronic myocarditis. But beyond the possibility of that I do not see that we can argue any further on the basis of what we have.

Dr. J. H. Means: Could that have been acute pulmonary edema? He had extreme dyspnea suddenly.

Dr. Cabot: Yes, I think that is a good suggestion. One does not ordinarily make that diagnosis unless they say he brought up a good deal of pinky fluid.

Dr. Means: We do see such cases though. We saw one the other day that did not bring up much fluid. We could tell that it was pulmonary edema from his chest. It filled acutely with râles. That is just the story of this case.

Dr. Maurice Fremont-Smith: How often does infarction occur without pain?

Dr. Cabot: It does occur. Only yesterday at the Brigham Hospital I discussed such a case, a case where I did not in any way suspect infarction. There was no pain, and then at necropsy a big infarct was found. Dr. Christian said they had had a number like that, and we have had at this hospital several others. Sometimes it comes in the course of a chronic disease like diabetes. A person gets comatose; it may be that if he were not comatose the pain would show up. But certainly we do see painless infarct, and this man may have it for all I know.

I suppose that sudden attack may have been pulmonary embolism. People do have pulmonary embolism and get over it. But I think Dr. Means's suggestion is better.

Dr. Means: I was rather interested at Rochester the other day to find that they were going to the expense of putting up two oxygen chambers in which they expected to put many of their pulmonary embolism cases. They thought that in a good many of the thyroid patients after operation there was pulmonary embolism, and if they put them into a chamber immediately they began to get cyanotic or dyspneic, they might save some that they are now losing. I think it is worth trying.

Dr. Cabot: It sounds reasonable.

Dr. Maurice Fremont-Smith: If a person has a pulmonary embolism large enough to cause marked dyspnea and require oxygen, is that the type that is going to get better anyway?

Dr. Means: Some do get well and some die. Therefore there is a group it is just touch-and-go with, and those are the ones they hope to save.

Dr. Cabot: Physiologically one would say, if oxygen is ever going to save, those are the ones.

Dr. Means: Of course if part of the lung is occluded, with no air entering, and if in the rest of the lung there is air entering normally, it is difficult to see what oxygen can do. Still I suppose usually there is a good deal of edema, and the oxygen might help overcome that.

Dr. Cabot: My mind goes back to one case of brilliant therapeutics with oxygen. That was a case of pulmonary tuberculosis who had just had the type of hemorrhage which, as necropsy showed, had gone soaking down into the lung without coming out. So that he did have a lot of his lung area cut off by blood. He was fighting and gasping in a sort of agony that I have never seen beaten. We got the oxygen and he went within a few minutes into complete comfort. He died a few days later of the miliary form of tuberculosis, which had been spread apparently by this hemorrhage. That is the most brilliant example of the use of oxygen that I have ever seen.

Dr. Means: I am certain that it is a very valuable drug, and the reason why we have had such poor results is that our method of administration is poor. We must give it with a mask or in a chamber to get results.

A Physician: Is death from pulmonary embolism due to asphyxia?

Dr. Cabot: So far as I know.

A Physician: What proportion of the lung would have to be closed off to be sufficient?

Dr. Cabot: I cannot answer that. That is a perfectly determinable fact, I should think, but not determined so far as I know.

I am asking myself the question whether this man has a nephritis. He has had some vomiting which may be accounted for by too much digitalis. We are not at all sure that it is. But there is nothing, it seems to me, in the urine that backs up the idea of marked damage to the kidney. I should suppose that if he is an arteriosclerotic, as I assume, the kidney will show some arteriosclerosis, but will not show that degree of destruction that we call nephritis.

This is the kind of case where we see intracardiac thrombi and their results. I should not be surprised if there were some such results, but I cannot put my finger on anything.

Dr. Means: With regard to hypertension: If this were a case of hypertensive heart disease with low pressure now from cardiac failure, one would expect a smaller pulse pressure.

Dr. Cabot: I think that question brings up a number of mysteries. One is the giving out of a heart with high blood pressure maintained. We see constantly people who die of obvious cardiac failure with stasis and the pressure is elevated up to death.

Dr. Means: The high pressure stasis of Elliott.

Dr. Cabot: We cannot say that the heart gives out, because it is up against too much and then the pressure goes down.

Dr. Means: I think the pulse pressure narrows in those cases.

Dr. Cabot: I would not say it does not narrow, but certainly it does not get down below normal in many cases of the type.

Dr. Means: Perhaps it is not smaller than normal actually but is relatively.

Dr. Cabot: Most of these arteriosclerotics have a high pulse pressure, 80 to 90 or 100. I do not know enough to say that it does not materially reduce that.

Dr. Means: Pulse pressure is dependent on the amount of blood thrown out per systole and diastolic pressure on the resistance at the periphery.

Dr. Cabot: The first thing to account for is why we have these two classes of cases, one that maintains high pressure to the end, and one that does not,—one in which the pressures are both low and one in which they are both high.

Dr. Means: The degree of congestion of the kidney may have something to do with it.

A Physician: Is the age a factor in these cases?

Dr. Cabot: Not so far as I know. I have seen it at various ages.

A Physician: How about the kidneys?

Dr. Cabot: It may be as Dr. Means suggests, that the amount of passive congestion has something to do with it. I have not observed this if it is so. I am discussing hypertension without nephritis of course. One certainly does see two groups of cases, those that have hypertension and keep it to death, and those that have hypertension and it falls; I do not know the difference.

This is the sort of case where one always asks the question about myocardial changes, and wishes he had something more definite to bank on. I do not think we have any evidence to say that he has myocarditis. I cannot say that he has it.

A Physician: But he has fibrillation.

Dr. Cabot: He has that, but of course we get that often without myocardial change. We see it in pneumonia where we cannot in any way link it up, and in thyroid troubles. It is intoxication we have to say.

Dr. Maurice Fremont-Smith: Was there too much digitalis?

Dr. Cabot: I should suppose so, yes.

Dr. Fremont-Smith: We cannot tell anything about the inverted T wave and P_2 interval. That should have been used as evidence that he did not have too much.

Dr. Cabot: The best I can say here is: a hypertrophied and dilated heart, probably with some arteriosclerosis, without valve lesion, possibly with changes acute or chronic in the heart wall, infarction and chronic myocarditis. Outside the heart he should show passive congestion and nothing else.

A Physician: Will there be pneumonia?

Dr. Cabot: That is a good suggestion, with his temperature. I had forgotten the temperature. We ought to consider what possibilities occur to us with this mounting temperature. He certainly did not show much in the way of symptoms of pneumonia, but that does not rule it out. This is just the sort of case that often shows an acute pericarditis at the end. But he did not have any physical signs of that.

Dr. Fremont-Smith: Do we have that temperature from infarct alone?

Dr. Cabot: Dr. Wearn tried to erect the theory that leucocytosis and fever could be explained by cardiac infarct. But certainly there are plenty of infarct cases where that does not occur.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Arteriosclerotic heart disease with congestive failure.
Terminal bronchopneumonia.
Embolic coronary thrombosis.

DR. RICHARD C. CABOT'S DIAGNOSIS

Hypertensive heart disease.
Hypertrophy and dilatation of the heart.
Arteriosclerosis.
Myocardial infarction? Myocarditis?
Chronic passive congestion.
Terminal pneumonia?

ANATOMICAL DIAGNOSIS

1. *Primary fatal lesions*

(Primary hypertension.)
Hypertrophy and dilatation of the heart.
Mural thrombi of right auricular appendix.

2. *Secondary or terminal lesions*

Infarcts of the right lung.
Chronic passive congestion, general.
Anasarca.
Hydrothorax, right.
Slight hydropericardium.
Ascites.

3. *Historical landmarks*

Obsolete tuberculosis of a bronchial lymphatic gland.
Slight chronic perisplenitis.
Arteriosclerosis.

Dr. Richardson: He was a stout man with edema of the lower extremities. The hands and the dependent portions of the trunk were puffy. There was 500 c.c. of fluid in the peritoneal cavity,—slight ascites. The appendix and gastrointestinal tract were out of the picture except for well marked chronic passive congestion,—velvety bluish-brown-red juicy mucosa. The liver was seven and a half cm. below the costal border. The diaphragm on the right was at the fifth interspace, on the left at the sixth rib.

In the right pleural cavity, 2000 c.c. of thin pale clear fluid, in the left nothing. There were a few pleural adhesions on the right and a few scattered ones on the left. Marked hydrothorax on the right, none at all on the left.

The trachea and bronchi contained much brownish-red fluid, indicating congestion of the lungs as the source, with one bronchial gland showing obsolete tuberculosis.

The left lung showed well-marked chronic passive congestion. The right lung showed well marked chronic passive congestion, and in the region of the middle and lower lobes several frank infarcts up to six cm. across. In small branches of the pulmonary artery leading to these infarcts, plugs, and in the right auricular appendix mural thrombi from which bits swept off into the pulmonary artery and branches. Of course the condition of chronic passive congestion is a favorable one for infarcts.

The pericardium contained 200 c.c. of thin pale fluid,—slight excess. The heart weighed 640 grams, which for him was about 300 grams, to say the least, in excess. There was a good myocardium macroscopically and microscopically. The coronary arteries were free, capacious, and showed only a slight amount of arteriosclerosis. In other words, there was a good aorta and branches with a hypertrophied heart. The valves were frankly negative. There was much dilatation on the left and marked dilatation on the right. The circumferences of the valves were a little increased: the mitral 11.5, the aortic 7.5, the tricuspid 14.5, the pulmonary 8 cm.

The liver showed the nutmeg markings of chronic passive congestion. The spleen weighed 393 grams, was moderately enlarged, showing dark brown-red elastic tissue typical of chronic passive congestion.

The kidneys weighed 465 grams. That is a large weight for the kidneys. They showed well marked chronic passive congestion; that is, the capsules stripped, the surfaces were bluish brown-red, there was a rather wide cortex, engorged vessels,—a typical picture.

The myocardium and kidneys macroscopically and microscopically showed only chronic passive congestion in the case of the kidneys and nothing to record in the case of the myocardium.

The prostate was slightly enlarged, negative.

Dr. Cabot: Dr. Means, as we look back from that necropsy, trying to reconstruct his illness with no nephritis and not much arteriosclerosis, have we any better guess than that he had a chronic hypertension and a big heart corresponding to it?

Dr. Means: There does not seem to be any better explanation.

Dr. Cabot: Do we know any other alternative when we find these big hearts? The man did not have hypertension under our observation. He has a big heart and nothing to account for it. Are we right in inferring therefore he had hypertension earlier?

Dr. Means: It would be very instructive to see if we could get in touch with his doctor and learn if anyone ever took his blood pressure before this.*

A PHYSICIAN: In cases where there is only slight arteriosclerosis of the larger vessels do you know whether or not there may sometimes be sclerosis of the smaller vessels?

DR. RICHARDSON: Anything is possible in arteriosclerosis. But if it is only slight in the aorta, and I examine all the great branches of the aorta below the diaphragm and above,—and usually it is noted clinically whether the radials and brachials are noticeable,—I think the chances are slight, although it is a possibility.

DR. MEANS: Did you see in the histology of the various organs any arteriosclerosis in the small vessels?

DR. RICHARDSON: No.

CASE 11482

MEDICAL DEPARTMENT

A married Swedish woman forty-six years old was referred from the Out-Patient Department June 27 complaining of weakness. Her mental condition was not normal and the history is not accurate. Her father died of tuberculosis. In childhood she had scarlet fever, brain fever and earaches. At twenty-six she had pleurisy and pneumonia. She had been married three times. By her first husband she had two children followed by a miscarriage. This husband she divorced. At one time she had vaginal discharge and burning micturition. At twenty-eight one ovary and one tube were removed. Two years later the cervix was amputated. That year she had two convulsions in which she was rigid all over, lost consciousness and bit her tongue. At this time she weighed 168 pounds, her best weight. She usually weighed 153.

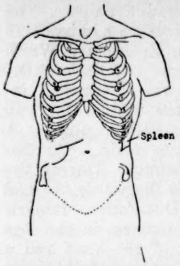
Records of the Out-Patient Department show that she came in December, six years before admission, complaining of staggering, shakiness and a rash on the hand of two years' duration. She denied venereal disease, but thought both her husbands had had blood disease. Examination showed the pupils slightly sluggish to light, and a typical papular serpiginous syphilitic eruption on the right palm extending up the wrist. A Wassermann was strongly positive. The blood pressure was 125/75. Under KI and mercury ointment the lesion rapidly healed. She was given six doses of arsphenamin and five of gray oil, later followed by six doses of neoarsphenamin and fifteen of gray oil. The treatment was continued to September, a year and nine months after her first visit. At that time she had shown negative Wassermann for seven months. During the treatment she complained of numbness of the abdomen, but no shooting pains. The following May, three years before admission to the wards, she came to the Emergency Ward. She was later referred to the Out-Patient Department, where a history was obtained of intermittent bleeding from the rectum for two years, usually slight, once half a pint. A diagnosis of hemorrhoids was made. At this time her weight was 149 pounds. During the year before her admission to the wards she had several times been in the Out-Patient Department with complaints of numbness in the legs and feet, pain in the back of the head and a sensation in the right foot as of walking on sand. In April, fourteen months before her admission to the wards, examination in the Nerve Room showed a positive Romberg. The pupils were normal. Finger to nose test was normal. The knee-jerks and ankle-jerks were not obtained. A lumbar puncture gave clear colorless fluid with a pressure of 125 to 290 to 160, Wassermann negative, total protein 26, goldsol 0000000000. A blood Wassermann was negative. Recalling her fourteen months later a house officer believed that her mental condition at this visit was not normal. In February, four months before admission, the hemoglobin was 40 per cent., the red count 1,300,000, the leucocytes 3,000.

The January before admission she had "grippe." Since that time she had been very weak. The last of January she was given some medicine at this hospital and was told to return in a month. About three weeks later her heart became irregular and her left leg became swollen. She came to the Emergency Ward very ill and was kept there for about a week. Later she was taken to another hospital, where she was, she believed, transfused without relief. Later she had all her teeth out. For the past few months she had felt fairly well and was able to go up and down stairs. Recently she had become weak again.

Examination showed a very pale, dyspneic woman in some respiratory distress. The teeth were false. The tongue showed marked atrophy of the papillae. It protruded in the median line with fine tremor. The apex impulse of the heart was felt 8 cm. to the left of midsternum, .5 cm. outside the midclavicular line, coinciding with the left border of dullness. The right border of dullness was 2 cm. to the right, the supracardiac dullness 5.5 cm. The action was rapid and regular. A marked systolic murmur was best heard at the pulmonic area. The pulses and arteries were normal. The blood pressure was 112/65 to 90/50. The lungs were clear. The abdomen was rather difficult to palpate. The spleen was felt. The liver edge was questionable (see

*Dr. William Otis Faxon, who saw the patient during the week before he entered the hospital, remembers the blood pressure at that time as between 140 and 150 systolic, 85 diastolic. We have been unable to obtain a report from any physician who saw the case earlier.

diagram). The pupils were normal. The knee-jerks and ankle-jerks were not obtained.



The temperature was 97° to 101.7°, the pulse 74 to 122 with a terminal rise to 151. The urine was pale and cloudy at one of six examinations, the specific gravity 1.010 to 1.014, the slightest possible trace of albumin at three examinations, no sugar, leucocytes at five, from ten per high power field to a sediment loaded with leucocytes. The blood showed 1,600 to 3,100 leucocytes, 66 to 30 per cent. polynuclears, hemoglobin 35 to 15 per cent., 750,000 reds, at entrance, afterwards 1,660,000 (after transfusion) to 488,000. The smear at entrance showed marked variation in size and shape, very many tailed cells, scarcely a normal cell, many large red blood cells, color index about 1, slight polychromatophilia; several fields showed macrocytes, microcytes, stippling and reduced platelets. June 28 the smear was similar and showed also diffuse basophilia, ring bodies of Cabot and occasional megaloblasts and normoblasts. Two later smears showed practically the same condition. July 24 there were approximately 1 per cent. of reticulated cells. A Wassermann was negative. The stools were negative.

July 1 the patient was reported pulseless three times. Two possible donors for transfusion proved to have blood incompatible with hers. That night the pulse was 120, the blood pressure 100/40, and the patient very weak. Next day transfusion of 500 c.c. of blood was done, with marked improvement in the general condition. The red count rose from 564,000 to 1,320,000, but fell rapidly, reaching 850,000 July 9. July 13 another transfusion of 500 c.c. was done. The red count rose to 1,660,000. The patient was in good spirits and seemed to be improving. July 30 however her condition was very low. No donors were available. July 31 she was practically comatose. The red count was 488,000. August 1 she died.

DISCUSSION

BY DR. MAURICE FREMONT-SMITH

NOTES ON THE HISTORY

All we can say from this history is that there was every opportunity for venereal infection, possibly of both types. The convulsion coming on in adult life for the first time, if it was the first time, suggests syphilis of the central nervous system. It also brings up the possibility of brain tumor. Sometimes the first sign of brain tumor will be a convulsion coming out of a clear sky in an adult.

A lesion on the palm is very suggestive of late syphilis, just as are papules in the creases about the mouth.

"Serpiginous" here probably refers incorrectly to the outline of the lesion. The real meaning of serpiginous is a lesion that is healing in one place and advancing in another, like the motion of a serpent.

She had pretty efficient treatment for syphilis, although no case of syphilis is sufficiently treated by two courses of arsphenamin. Treatment should certainly extend over three or four years. They do not seem to have made any special effort to treat a neurosyphilis or to find out whether a neurosyphilis really existed or not. With the history of convulsion and with the abnormality of the pupils a lumbar puncture was certainly indicated; because if she had neurosyphilis it would change one's feeling as to treatment. Here after the lesion cleared up and a couple of courses of arsphenamin had been given they felt that they had done enough,—or else she did not return for further treatment.

The sensation of "walking on sand" again suggests neurosyphilis. That the pupils are normal is an interesting observation, because abnormal pupils are frequently the first sign of neurosyphilis. Knee-jerks are rather frequently normal in tabetics, the ankle-jerks less frequently normal, but still normal often enough. The pupils are rarely normal in neurosyphilis.

Did she have at one time a positive spinal fluid? Stokes has shown that a great many cases of secondary syphilis show abnormal spinal fluids, some of the cases returning to normal without ever giving symptoms of tabes or paresis. It seems a reasonable assumption that in a generalized disease like syphilis the spinal fluid should be abnormal at some time. One would however expect to find some changes left. It is rather rarely that an injured central nervous system will later give a fluid absolutely normal. I should expect the protein at least to be up if she had had an old neurosyphilis.

On the other hand the absent knee-jerks and ankle-jerks, the positive Romberg, the shooting pains in the legs, the numbness of the feet make a picture that is very hard to explain on any other basis. A normal fluid does rarely occur in neurosyphilis.

On the basis of this meager blood examination one would suspect a pernicious anemia, chiefly from the low leucocyte count. That brings up the possible relationship of syphilis to pernicious anemia. Given a case with a blood smear typical of pernicious anemia, with a low white count and low platelets,—can that blood picture be due to syphilis? Cases of anemia indistinguishable from pernicious anemia have been reported as due to syphilis. Suppose in such a case one find a positive blood Wassermann in the serum. Should one then feel that syphilis is present, and if present is responsible for the so-called pernicious anemia? Of course we have to remember that there are certain non-syphilitic conditions in which positive Wassermanns occur. Yaws is such a disease. In jaundice false positives may occur. In a case of pernicious anemia we have excess bile pigments in the serum. When we look at the blood serum we find that it is yellow, due to abnormal hemolysis of the red cells. Could we not then say that a positive Wassermann in a case of pernicious anemia might well be due to excessive bile pigments in the serum, and not to syphilis? Is there any relation between this woman's syphilis and a blood condition which looks like pernicious anemia? An unusually high incidence of positive Wassermanns does not occur in pernicious anemia, however.

If she had pernicious anemia the extraction of teeth was a move of doubtful experiency. She might have had very severe hemorrhage from it; moreover we could hardly expect any benefit.

Her improved condition during the past four months may well have been a marked remission.

NOTES ON THE PHYSICAL EXAMINATION

We have nothing to suggest any disease of the heart. The murmur is well explained by the profound anemia.

The pupils were normal at the second observation.

Of course we have to remember that knee-jerks will disappear permanently after the peripheral neuritis following diphtheria. Some normal individuals have no knee-jerks that can be elicited. The ankle-jerks however are much more constantly found. Sciatica will temporarily disturb the ankle jerk; but absent ankle jerks have to be looked upon as a very important sign that the reflex path is disturbed.

Another point that we must consider is this: Here is a woman in whom we suspect pernicious anemia. We know that in pernicious anemia very definite cord changes occur. Sometimes those cord changes occur before any abnormality can be found in the blood. The cord changes are in the nature of a degeneration of the posterior tracts and sometimes of the lateral tracts, then giving us what is known as combined system disease. The first sign of posterior tract degeneration is difficulty with the deep sensorium. Toe-position sense is very apt to go first, vibratory sense (tuning fork) early. Later on come the signs of ataxia. A very definite ataxia may be due to pernicious anemia. That might well explain her staggering. It would explain the numbness. It would explain the absent knee and ankle jerks, and possibly this is the entire explanation for the symptoms which at first suggested central nervous system syphilis. The important thing to remember here is that those nerve signs and symptoms may occur as the first symptom of pernicious anemia, may occur before any changes in the blood can be found at all.

She probably developed a pyelitis.

There is evidence of a very marked aplasia of the bone marrow. It might be aplastic anemia secondary to arsenic or benzol poisoning. Secondary aplastic and primary anemia are the only two conditions I know of that give as low a white count as this.

A PHYSICIAN: Could this be due to fish tapeworm?

DR. FREMONT-SMITH: The white count is usually not low in the anemia caused by dibothriocephalus; also the tendency to production of large erythrocyte forms is not so marked.

A PHYSICIAN: There is the leukopenia of Banti's disease.

DR. FREMONT-SMITH: Is there? I did not know that. Of course we get a leukopenia in measles. Does it ever go as low as this?

A PHYSICIAN: How low does it go in typhoid fever?

DR. FREMONT-SMITH: Not usually below three or four thousand I should say.

DR. MORRISON: In sepsis without any resistance it is very low.

DR. FREMONT-SMITH: That is again three to five thousand.

DR. MORRISON: I have seen it as low as two thousand.

A PHYSICIAN: Could it be subacute endocarditis?

DR. FREMONT-SMITH: In subacute endocarditis there are often very marked changes in white count. It varies even as one watches the patient, in the same way that the showers of endothelial leucocytes may come out into the blood and disappear rapidly.

DIFFERENTIAL DIAGNOSIS

Let us stop a moment and think of the various conditions which could give us a blood picture similar to this. I mentioned the possibility of syphilis giving a picture like pernicious anemia. Dibothriocephalus gives a picture sometimes extremely like the picture of pernicious anemia. In those countries, however, where dibothriocephalus tapeworm infection is very frequent the percentage of pernicious-anemia-like blood pictures is extremely low. Dr. Wearn told me the other day that he had seen the blood picture of pernicious anemia in a case of pulmonary tuberculosis. Of course one did not know that that was not a combination of tuberculosis plus pernicious anemia. It certainly is very rare in tuberculosis.

We see pictures that suggest pernicious anemia in carcinoma of the stomach. A few years ago Mann at the Mayo Clinic did complete gastrectomies on dogs and found that they developed a blood picture very similar to pernicious anemia.

Sprue has to be thought of. It is not clear whether there is such a disease. Wood says that whether one makes a diagnosis of sprue or pernicious anemia depends on whether the patient is in the tropics or in the temperate zone. Sprue however, if there be such a separate disease—and I think there is—can have a blood picture indistinguishable from pernicious anemia, and very rarely in sprue one has the cord degeneration that is frequent in pernicious anemia. In sprue also the hydrochloric acid is absent or very low in the stomach. But the neutral red test seems to be specific for pernicious anemia. When neutral red is injected intramuscularly, in every condition so far as we know except pernicious anemia it appears in the stomach contents. It appears in the stomach contents in carcinoma of the stomach even when the hydrochloric acid is absent, according to the work of Davidson at the City Hospital.

I can think of no other conditions that could be confused.

Why the repeated transfusion? Possibly they were hoping that by transfusion the marrow might be stimulated, so that the blood count would rise again perhaps for several months before going down. I am not at all sure that our work here has substantiated that belief. There are a great many cases transfused in which there is absolutely no increase in the bone marrow activity. Personally I feel about blood transfusion that it is entirely a psychological question and should be decided entirely on psychological grounds. If the patient and the family want life prolonged, repeated transfusions will do it—twelve or thirteen times—as long as we keep up the transfusions. As for any definite benefit, I have not seen any.

Shall we find any evidence of syphilis at necropsy? Very often we believe clinically the patient has syphilis and at necropsy Dr. Richardson tells us there is nothing.

Is there possibly an old healed tuberculosis, with the history of her father and the history of pleurisy?

What relation if any is there between the undoubted syphilitic infection and the pernicious anemia? Shall we find anything in the bone marrow other than the usual typical picture of aplasia in pernicious anemia?

A PHYSICIAN: Do we get an aplastic anemia from the effects of arsphenamin?

DR. FREMONT-SMITH: I believe one can.

DR. JOHN S. LAWRENCE: The nearest I have seen was a purpura.

DR. FREMONT-SMITH: I have seen one case I felt sure was definitely due to arsphenamin therapy. But I cannot back it up.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORDS)

Pernicious anemia.
Transfusion.

DR. MAURICE FREMONT-SMITH'S DIAGNOSIS

Pernicious anemia.
Syphilis?

ANATOMICAL DIAGNOSIS

1. *Primary fatal lesion*

   Pernicious anemia.

2. *Secondary or terminal lesions*

   Hyperplasia of the bone marrow.
   Slight chronic endocarditis of the mitral valve.
   Hypertrophy and dilatation of the heart.
   Fatty degeneration of the myocardium.

Passive congestion of the lungs.
Atrophy of the mucosa of the stomach.
Hydropericardium.

3. *Historical landmarks*

Chronic pleuritis.
Obsolete tuberculosis of a bronchial gland.
Foci of obsolete tuberculosis of apices of lungs.
Scar of old operation wound.

Dr. Richardson: Unfortunately we could not examine the head, so I cannot say anything about the neurosyphilis. It was a typical picture of pernicious anemia, and we remember well enough that in pernicious anemia degeneration of the cord and of the brain may occur.

She was fairly nourished. The skin was a pale old ivory color, the fat lemon-yellow. The legs, ankles and feet were slightly swollen and pitted sightly. There was an old linear scar from the pubes upward. The muscles were pale.

The stomach was rather small, the mucosa flat, smooth and of a pale brownish-yellow color. There was at one time an attempt to connect pernicious anemia with changes in the mucosa of the stomach, but that has been abandoned I think.

The intestines were negative except that here and there in the mucosa were small hemorrhagic areas.

The bronchial glands were slightly enlarged and in one of them there was some obsolete tuberculosis. The apices of the lungs also showed small masses of obsolete tuberculosis. The lungs otherwise showed considerable edema.

The pericardium contained 200 c.c. of thin pale clear fluid. Here again in pernicious anemia there was a hypertrophied and dilated heart, the heart in this case weighing 430 grams. If her heart had weighed 230 grams we should have passed it by as all right. The myocardium was of good consistence, pale brown-red, and the endocardium in the region of the left ventricle mottled with minute to small pale areas,—the so-called tiger-lily heart, the fatty degeneration associated with pernicious anemia. The cavities on the left were full sized; slight dilatation on the right. The valves were frankly negative except that the mitral valve along the free margin at one place showed a very small fibrous columnar mass—a little patch of chronic endocarditis. It was there and has to be mentioned, but of course had no effect one way or another on the welfare of the individual.

The liver weighed 1905 grams, rather large. There was some vacuolation of liver cells. Otherwise it was frankly negative. The spleen weighed 444 grams, moderately enlarged, the tissue plump. The microscope showed a few myeloid cells here and there. The left tube and ovary and the right tube were wanting. There was a small slender band of adhesions extending from the region of the right ovarian ligament to the wall of the intestine, but it did not constrict the intestine to any appreciable extent.

The blood generally was rather pale, with very little clotting. The bone marrow was pinkish brown and a little softer than usual. Of course in typical pernicious anemia, especially in the femur, the marrow is dark raspberry red. We were not allowed to go into the femur, so I cannot speak about it.

Dr. Fremont-Smith: Is the incidence of gall-stones in pernicious anemia higher than we find in normals?

Dr. Richardson: I think it is not.

Dr. Fremont-Smith: There is so much more bile pigment in the blood all the time.

Dr. Richardson: I mentioned the bile here as being thick, but there were no gall-stones.

CASE 11483

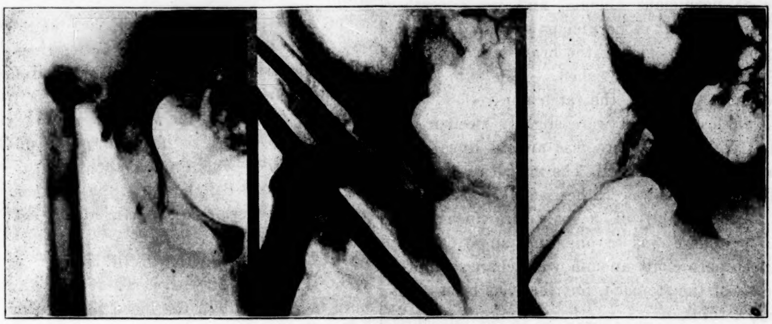
Orthopedic Department

A Turkish tailor of seventeen was admitted December 3. He gave a history of his father's dropping him when he was a baby. There was no trouble until he began to walk.

Examination showed three inch shortening on the left. The greater trochanter was prominent just below the crest of the ilium. There was decided rotating motion in walking. Positive Trendelenberg. Movements in all directions except hyperextension. Abduction of 25°. The left leg was much smaller than the right. X-ray showed the condition as in Plate I.

A diagnosis of single congenital dislocation of the left hip was made.

Operation was done December 7, open reduction of the dislocation. Incision was made over the left hip exposing the capsule of the joint, which was elongated, hour-glass shaped, the head of the femur resting on the ilium two and a half inches above the true acetabulum. The capsule, which was very thick and tough, was separated anteriorly and the head of the femur was manipulated out of the sac. The entire attachments of the muscles of the greater trochanter were separated subperiosteally. The lesser trochanter was separated entirely with a chisel, thus freeing the attachment of the psoas. This mobilized the hip, and it was possible to discover the true acetabulum, which was filled with dense fibrous tissue. This was in large part removed. The acetabulum was of good depth and had a strong upper shelf. The wound was closed. Ice tongs were inserted into the lower end of the femur just above the condyles. Fifty pounds traction applied.



CASE 11483. Single congenital dislocation of the left hip in a young adult, reduced by open operation.

PLATE I. Condition before operation. Two and a half inches actual shortening.

PLATE II. Head of femur approaching acetabulum through skeletal traction exerted on its lower end by "ice tongs."

PLATE III. The reduced head of the femur occupying the acetabulum.

Treatment was continued for four weeks, skeletal traction of forty to fifty pounds. X-rays (see Plate II) taken after three weeks showed the head of the femur approaching the level of the acetabulum.

January 9 the old incision and scar were excised and the hip joint exposed in the Smith-Petersen manner. The head of the femur was found to be about an inch and a half above the acetabulum. The structures about it were loosened up and the acetabulum cleared of its contents, old fibrous tissue. By internal rotation and abduction the head of the femur was placed in the acetabulum. The wound was closed. A double plaster of Paris spica was applied, holding the left lower extremity with the hip reduced.

After three weeks in plaster the patient began to walk.

Discussion

BY DR. NATHANIEL ALLISON

Single congenital dislocation of the hip in individuals who have pasesd their fifteenth year presents a serious surgical picture. The difficulties encountered in attempts at reduction when childhood is passed are very great and increase with each year of development, so that they may be almost insurmountable when the patient has reached or passed adolescence.

The patient on whom these operations were done was a healthy young male adult and it seemed advisable to have him undergo the rather severe treatment required in order that he might walk without his distressing limp, and in order that he might cease to be, in a sense, a crippled individual.

The difficulties of reducing congenital dislocations in older individuals are very great. The difficulties in early childhood are not great. Early diagnosis and early treatment are necessary for successful reduction in these cases.

SUBSEQUENT HISTORY

At the end of three months the patient was able to return to work as a tailor and has since been actively engaged in making his living. The left hip has caused him no sense of disability since that time. He walks upstairs easily, sits in a chair comfortably, and has difficulty only in putting on his left shoe. The left hip is stiff, in a position of twenty degrees of flexion, ten degrees of abduction and slight external rotation.

DIAGNOSIS

Single congenital dislocation of the left hip.

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CHANGING VALUES IN MEDICINE

The awakening interest of our medical societies in the dissemination of information to the public is but an indication of the trend of the times towards increasing frankness with the public on the part of those who have assumed more or less responsibility for its physical welfare. Public health matters and the prevention of disease are absorbing more and more of the interest of intelligent physicians, and intelligent physicians are becoming more and more eager to impart this information to their lay brethren. The voodoo of the witch doctor and the mysterious pomposity of the medicine of a later date are vanishing before the refreshing coöperation of modern health methods. Knowing more than ever before we are more willing than ever to admit our shortcomings and we believe that the public appreciate honesty and an open mind.

Many attempts at medical education of the layman are being made by public and private enterprise, and the banker or the carpenter who does not know how to prevent smallpox and typhoid fever and diphtheria has done little reading as he ran.

Among many other efforts may be listed the educational campaign of the Massachusetts General Hospital, which for almost a year has published each week in a Boston Sunday newspaper an authoritative health article by some member of the staff; the health examination campaign of the Boston Dispensary, and the persistent endeavors of our state, city and federal health services to awaken in both physician and layman an appreciation of the importance of health preservation. The practicing medical profession is, of course, unable to advertise its services in any specific or individual sense, and consequently the public is much more familiar with the periodic physical examination as offered by the Life Extension Institute than it is with the same type of service as performed by the family physicians.

There is naturally a marked contrast between such activities and those of the beloved and late lamented old country practitioner with his Goddard buggy and his sorrel mare, hitched at one time or another to every gate post in the town while her driver dispensed calomel and comfort to the sufferer within. In the words of a famous French clinician, it was his lot to cure rarely, to alleviate often, to console always. Pasteur, the greatest of humanitarian scientists, whose centenary we have recently observed, spelled the doom of old style practice with the discovery of the bacterial origin of many diseases, for the Art of Medicine became tempered and strengthened by the Science of Medicine, and the automobile drove the sorrel from the road. God rest her bones! She deserves to be canonized in some equine heaven for the peace of mind she has brought to many a lonely farmhouse.

Science has indeed entered the service of modern medicine, but she will never entirely replace the Art of practice, for minds as well as bodies must be ministered to and judgment cannot be calibrated nor can common sense be sublimated in a test tube. The muscle of a heart can be made to write its record on a photographic plate, but the electrocardiogram cannot read the suffering in a human eye or speak the words that cause that look to disappear. The late Sir James MacKenzie knew more about the human heart than any man, but before he died he warned his colleagues that Science must be not the mistress but the handmaiden of Medicine.

Pandora's box is open and its plague of ills have flown abroad, and presumably sickness will be one of the troubles of mankind until its goal is reached. Nevertheless, while the Art of Medicine must always be employed to soften these sufferings, its Science can do much to prevent them, and its final limitations are by no means yet in sight.

Prevention is becoming the keynote of modern medicine although its onward movement is slow, partly hindered by a natural distrust of anything that seems to be new; hindered more than we realize by the anti-social activities of those apparently well meaning groups of individuals who are opposed to animal experimentation, to vaccination, to the Schick test and to the medical profession in general; hindered by the cultists who cannot always be dismissed from mind at will; hindered by legislation which permits the insufficiently trained to practice medicine, and hindered by the shamefully inadequate salaries which are meted out to public health officials.

In a broad sense the public is the keeper of its own health, but only by education can it be made to realize this fact, and only those who have attempted the education of the public know what a stupendous task it is. Many diseases cannot be prevented; many individuals, with the best of care, will become sick and will die of unpreventable diseases, but it is possible definitely to prevent many diseases and it is possible so to live that one's chances of acquiring many other diseases are materially lessened. Providing legislation that will permit only the well qualified to practice, and providing salaries that will attract a high type of individual into public health service, and paying attention to the advice offered are the means by which this end may be attained.

The preservation of health by the employment of specific measures and by the annual stock-taking of personal health—the periodic health examination—provides a certain amount of insurance that must be acceptable to the intelligent man, for insurance is cheaper than standing losses, and it is pleasant neither to undergo illness nor to die before one's allotted span is run.

It is pardonable and perhaps logical to hunt for the colored man among the cordwood, for it is not easy for the average, commercially efficient business man to understand why any class of workers should seek to ruin their own business and diminish their own returns. Fortunately it is not necessary to believe that such will be the case although there are various factors in the practice of medicine that render it different from any other type of professional service.

Granted that the living of the old style practitioner is derived indirectly from the misfortunes of his fellow beings, it is nevertheless not pleasant to view the sufferings of another, and most physicians would do away with suffering if it were in their power. Despite the firm convictions of a few who have had unfortunate experiences, the profession of medicine is essentially a humanitarian one, and its rewards cannot be measured on a purely economic basis.

It is not to be doubted that a wholly therapeutic type of practice will and must suffer from the wide dissemination of information on health matters in the same ratio that the physical well being of the community improves. The practitioner who cannot adapt himself to a changing order will find that he has less work to do, but few will not change, and change to their own advantage, when they find themselves faced with the necessity, for the world will roll on and we must roll with it, since gravitation prevents us from abandoning it entirely.

The transition must come from a well meant but comparatively ineffective type of practice to an equally well meant but far more effective type, in which each practitioner will be a private health official, working in harmony with an equally respected and efficient body of public health officials, all impressed with the same idea—that many of the physical calamities of life can be avoided.

Of course, it would be absurdly Utopian to believe that at one stroke the usefulness of therapeutic medicine will fall before the advance of preventive medicine. The surgeon will still hew to the line and the specialist will explore the crannies of that section of the body that has been allotted to him. And in this, our so-called temperate zone, the tonsils will still fall before the scythe of the reaper, though we hope in lesser numbers than before. But by and large many of our ailments will be prevented and he who swings along in the march will find that there is much for him to do, though of a different order.

REPORT OF THE THIRTY-SIXTH ANNUAL MEETING OF THE ASSOCIATION OF AMERICAN MEDICAL COLLEGES

The Thirty-Sixth Annual Meeting of the Association of American Medical Colleges was held at Charleston, S. C., on October 26, 27 and 28, 1925. The policy of visiting a medical school each year instead of meeting with the Council on Medical Education of the A. M. A. in Chicago, is to be continued, and the meeting next year will be in Cleveland, Ohio, at Western Reserve University.

Charleston is well known politically and socially as of unique historical interest. But the student of the history of medicine in America has found and will continue to find much that falls within his field. John Lining[1], Alexander Garden[2], Lionel Chalmers and David Ramsay are some of the worthies of earlier days.

The formal program for the meeting of the Association was devoted largely to the consideration of the technic of teaching. The Round Table discussions were very valuable and here "The Irregular Student" proved to be the most interesting topic. Irregularity may be due to illness, to special interest in some subject in the medical course or to financial embarrassment. Illness of the student presents no special administrative problem. The student with some special interest has in most cases unusual gifts, is the "superior student," and the problems are to give him suitable opportunity and to protect from undue handicap superior students who are financially embarrassed. The years in the medical school fall within the critical period of the life of most students of medicine, and poverty at this time may permanently stunt developing powers. Fellowships awarded on the basis of proved ability and permitting work along lines which are parallel with, if not a part of the medical course, offer one solution if the medical course has to be interrupted. Medical education is very expensive and should not be given to students of slight natural endowment.

There was given a brief report on the Commission on Medical Education. The Commission was appointed at the suggestion of the Association, and financed by the Association, the American Medical Association, the General Education Board, and the Carnegie Foundation. At its first meeting in Buffalo recently, President A. Lawrence Lowell of Harvard was elected Chairman, and Dr. W. C. Rappleye is Executive Secretary. The personnel of the Commission was announced some time ago and included medical educationalists and a number of men distinguished for their wide vision over the whole field of education. Medical education will be considered from every point of view, and information will be collected for an intensive study of some of the major or more urgent problems. One of these problems is how to educate the medical student today so that he will not be behind the times after twenty-five years of practice.

The greatest need of medical education at the present time is freedom so that it may be established on an experimental basis. An effort will therefore be made to persuade the Federation of State Boards to favor, and the individual State Boards to adopt, a policy or plan which will make experiment possible. The legal obstacles do not seem insuperable. At the present time such experiments are not possible without disqualification for licensure, in some states, of the graduates of the experimenting school. The Association voted to empower its executive council to permit experiments in medical education in any school which meets its approval. These are hints of better days to come.

REFERENCES

1 Philosophical Transactions, London, 1743-1744, Vol. XLII. "Extracts of Two Letters from Dr. John Lining, Physician at Charlestown in South Carolina, to James Jurin, M.D.F.R.S., giving an account of Statical Experiments made several times a day upon himself, for one whole year, accompanied with meteorological observations, to which are subjoined Six General tables deduced from the whole year's course."

2 Beginnings of Natural History in America. By George Brown Good. See page 80 for statement made by Alexander Garden.

THIS WEEK'S ISSUE

Contains an article entitled "Chemical Studies of the Blood in Pneumonia," prepared by:

Buckman, Thomas Ellwood, A.B., A.M., M.D., Harvard Medical School 1917. Previous to recent removal to Jacksonville, Fla., he was Instructor in Pediatrics, Harvard Medical School; Visiting Pediatrician, Boston City Hospital; Director, Blood Research Laboratory, Boston City Hospital; Visiting Physician, Massachusetts State Infirmary; Chemist, Robert Brigham Hospital, etc.; Member, American Pediatric Society, American Society for Clinical Investigation, etc. Associated with Dr. Buckman are

Adams, Frank Dennett, M.D., Harvard Medical School 1917,

Smith, Millard, M.D., Harvard Medical School 1923 and

Edwards, Mr. H. T., Research Chemist.

The Massachusetts Medical Society

NEW ADDRESSES

To Fellows of the Society:

Please send at once to the Secretary any changed addresses for the *Annual Directory*, soon to be issued.

W. L. Burrage, *Secretary.*

182 Walnut St.,
Brookline, Mass.

MEMBERSHIP CHANGES

1908 } Buckley, James Thomas, Worcester, 8 Loudon
1925 } Street. Restored by Council, October 7, 1925.

Dr. William W. McKibben has moved from Worcester (Worcester) to Miami, Florida (Non-Resident List), office 205 Calumet Building.

Dr. George Robbins has moved from South Boston (Suffolk) to Middleton (Essex South), Essex Sanatorium.

Dr. Samuel Tartakoff has moved from New Bedford (Bristol South) to Brighton (Middlesex South), 180 Washington Street.

Dr. Ray L. Whitney has moved from Cromwell, Conn. (Non-Resident List), to Waverley, Belmont, Mass. (Middlesex South), the McLean Hospital, P. O. Box 8.

MISCELLANY

THE CHRISTMAS SEAL SALE

Everything is in readiness for the opening of the Christmas Seal Campaign which will be conducted by the Massachusetts Tuberculosis League this week. Mr. Frank Kiernan, the Executive Secretary, has announced that demands from all of Massachusetts for seal materials exceeded that of 1924 by 30 per cent. It has been necessary for the Massachusetts

Tuberculosis League to scour the country for additional Christmas Seals, posters and other campaign material. Over 3,500,000 Seals are en route from the manufactuirng plant at Scranton to meet the eleventh hour demands.

Mr. Kiernan states that in spite of the fact that the consumption death rate has been reduced more than one half in recent years it still remains the principal cause of death during the ages of 19 and 40. This shows that still more adequate means must be provided to stamp out the disease. Of all the plagues that have afflicted the world none is more deadly than tuberculosis, the "*Great White Plague.*" Although all other plagues, ancient and modern, have swept a country or community for a few weeks, months or years, tuberculosis has seized its victims for over 2,500 years. It fights from ambush and strikes the rich, the poor, the brilliant, the stupid, the weak, the strong.

Dr. John B. Hawes, the President of the Boston Tuberculosis Association, states that this is the second year that the greater portion of the money has been raised through Christmas Seals by means of the letter sale. This means an immense amount of work, especially the preparing of an up-to-date mailing list of over 100,000 names. The first year's mailing list was very rough and inaccurate; this year's contains a list of those people who are really in sympathy with the work and does not bother those who are not interested. To show that the public has responded, these figures are of interest: Three years ago we raised from Christmas seals $14,000; two years ago, $25,000; last year $50,000. This shows more than anything else that the public is becoming more and more interested in the work and more sincerely endorses what is being done.

The Thurlow Advertising Service, Franklin P. Shumway Co., Hunt-Luce Agency, Barton, Durstine & Osborn, Richard L. Fenton, Conlon, Prescott & Co., Donnelly Advertising Co. and many other agencies are assisting in the publicity as well as all the Greater Boston newspapers.

NEGATIVE DIAGNOSIS IN PULMONARY TUBERCULOSIS

BY DR. H. H. WALKER, LEAHI HOME, OAHU

THE man who makes good diagnoses in Pulmonary Tuberculosis does so not because he is endowed with any divine foresight or unusual ability, but because he strictly follows two fundamental rules. The first of these is the careful accumulation of all relevant data, i. e., careful history of the case, appraisal of the patient's general condition, thorough painstaking physical examination always employing routine procedure, sputum examination, X-ray, accessory tests, and finally a correlation of all data obtained. The second fundamental is to be unduly cautious in making a negative diagnosis. The whole may be summed up by saying "Never hurry." When in doubt always reëxamine the patient, observing him closely in the meantime.

The negative diagnosis is always dangerous, and I believe it is just at this point where most physicians err. There are far more mistakes made in pronouncing the positive case to be negative than in pronouncing the negative to be positive. The reason for this is undoubtedly the fact that pulmonary tuberculosis is insidious in onset is lost sight of. It comes down to the old fable—the race between the hare and the tortoise. In diagnosing tuberculosis the slow-moving turtle will win every time.—*Queen's Hospital Bulletin.*

THE WORLD'S CHILDREN

Ground Plan of Model Playground

The Children's Bureau has on hand for free distribution a ground plan of its playground model. The model was prepared for the bureau as an illustration of how to use a lot of 5 acres or more as a recreation center for children.

MASSACHUSETTS MORTALITY STATISTICS: 1924

THE Department of Commerce announces that the 1924 death rate for Massachusetts was 1,211 per 100,000 population as compared with 1,295 in 1923. This decrease in 1924 is largely accounted for by decreases in the death rates from pneumonia, all forms (from 131 to 100 per 100,000 population), diseases of the heart (from 241 to 225 per 100,000 population), influenza (from 21 to 8), whooping cough (from 12 to 4) and measles (from 9 to 4).

An increase appears in 1924 in the death rate from cancer (from 118 in 1923 to 124 per 100,000 population).

Deaths in Massachusetts

Cause of death	Number		Rate per 100,000 estimated population	
	1924	1923	1924	1923
All causes[1]	49,358	52,166	1,210.5	1,295.2
Typhoid and paratyphoid fever	70	70	1.7	1.7
Malaria	1	1	[2]	[2]
Smallpox	2	—	[2]	—
Measles	171	344	4.2	8.5
Scarlet fever	161	160	3.9	4.0
Whooping cough	155	501	3.8	12.4
Diphtheria	529	586	13.0	14.5
Influenza	319	827	7.8	20.5
Dysentery	16	11	0.4	0.3
Erysipelas	109	116	2.7	2.9
Lethargic encephalitis	58	86	1.4	2.1
Meningococcus meningitis	62	59	1.5	1.5
Tuberculosis (all forms)	3,513	3,565	86.2	88.5
Of the respiratory system	2,981	3,079	73.1	76.4
Of the meninges, central nervous system	242	248	5.9	6.2
Other forms	290	238	7.1	5.9
Syphilis[3]	494	512	12.1	12.7

Cancer and other malignant tumors	5,052	4,760	123.9	118.2
Rheumatism	160	123	3.9	3.1
Pellagra	12	11	0.3	0.3
Diabetes mellitus	763	861	18.7	21.4
Meningitis (nonepidemic)	112	137	2.7	3.4
Cerebral hemorrhage and softening	4,618	4,546	113.3	112.9
Paralysis without specified cause	114	115	2.8	2.9
Diseases of the heart	9,178	9,701	225.1	240.9
Diseases of the arteries, atheroma, aneurysm, etc.	2,005	1,977	49.2	49.1
Bronchitis	357	485	8.8	12.0
Pneumonia (all forms)	4,075	5,274	99.9	130.9
Respiratory diseases other than pneumonia (all forms) and bronchitis	270	319	6.6	7.9
Diarrhea and enteritis (total)	1,011	1,109	24.8	27.6
Diarrhea and enteritis (under 2 years)	850	966	20.8	24.0
Diarrhea and enteritis (2 years and over)	161	143	3.9	3.6
Appendicitis and typhlitis	573	535	14.1	13.3
Hernia, intestinal obstruction	418	375	10.3	9.3
Cirrhosis of the liver	221	208	5.4	5.2
Nephritis	3,022	3,025	74.1	75.1
Puerperal septicemia	198	174	4.9	4.3
Puerperal causes other than puerperal septicemia	394	387	9.7	9.6
Congenital malformations and diseases of early infancy	3,372	3,478	82.7	86.4
Suicide	488	450	12.0	11.2
Homicide	109	110	2.7	2.7
Accidental and unspecified external causes (total)	2,849	2,828	69.9	70.2
Burns (conflagration excepted)	256	264	6.3	6.6
Accidental drowning	355	254	8.7	6.3
Accidental shooting	29	25	0.7	0.6
Accidental falls	703	665	17.2	16.5
Mine accidents	—	—	—	—
Machinery accidents	63	85	1.5	2.1
Railroad accidents	110	140	2.7	3.5
Street-car accidents	61	68	1.5	1.7
Automobile accidents[4]	685	611	16.8	15.2
Injuries by vehicles other than railroad cars, street cars, and automobiles[5]	52	76	1.3	1.9
Excessive heat (burns excepted)	13	20	0.3	0.5
Other external causes	522	620	12.8	15.4
All other defined causes	4,219	4,203	103.5	104.4
Unknown or ill-defined causes	108	137	2.6	3.4

[1] Exclusive of stillbirths.

[2] Less than one-tenth of 1 per 100,000 population.

[3] Includes tabes dorsalis (locomotor ataxia) and general paralysis of the insane.

[4] Does not include deaths from collisions with steam and street cars.

[5] Includes airplane, balloon, and motorcycle accidents.

THE OCTOBER MEETING OF THE MASSACHUSETTS ASSOCIATION OF BOARDS OF HEALTH

THIS meeting was held at the Twentieth Century Club, October 29, 1925. The subject considered was "What Activities Should Boards of Health Undertake?"

Exhibits by Dr. Chapin of Providence, Dr. Jones of Swampscott and Dr. Denney of Brookline, showing cards and literature published by health officials, were interesting and instructive.

Dr. Mahoney, Health Commissioner of Boston, moved that a committee be appointed to study forms in use by Boards of Health and report recommendations for standardization of these data. The motion was adopted. This matter will come up for further discussion at a subsequent meeting.

Dr. E. H. Trowbridge of the Worcester Department of Health delivered the following address:

WHAT ACTIVITIES SHOULD BOARDS OF HEALTH UNDERTAKE?

Mr. President, and Members of the Association:

In the brief time allotted to consider this subject, I know it would be an impossibility to enter into all the details of the activities of Boards of Health, and therefore it will be my intention to consider some of the major activities, hoping that the problems and activities of the Boards of Health of the various localities will be fully discussed.

It appears to me, that Boards of Health have a fourfold duty or function to perform, and for convenience may be classified as follows:

First: Every Board of Health owes its allegiance to the state.

Second: Every Board of Health owes fidelity and loyalty to its respective municipality.

Third: Every Board of Health should court a widespread coöperation and maintain mutual relations with the profession.

Fourth: Every Board of Health should be a bulwark of safety for the health of the people and the sanitary condition of their respective communities.

Under these four divisions we may consider "What activities should Boards of Health undertake?"

The state has wisely and judiciously enacted laws pertaining to the health and sanitation, and these laws are just as applicable to the people who reside on the shores of Cape Cod, or who dwell in the Metropolitan district, or who abide on the hills of the far away Berkshires.

The sole purpose and object of these laws is to conserve the health of the people of this state, and the machinery for executing these various laws is the Boards of Health.

It is very apparent that each district presents its own problems and difficulties; and the activities of the Board of Health of one district may be far more or less stringent than in another district or even in the adjacent district, and hence, these activities will be somewhat varied, although the essential principles of the preservation of health remain the same throughout the state.

Then too, the personnel of the various Boards of Health is an *all* important factor, for unless the members are thoroughly familiar with health matters and unless they devote time and thought and study to the duties of their department, then such Boards cannot function with any degree of honor to themselves, as a Board or with any degree of success in behalf of their respective communities.

Right here arises the all essential question, "How can Boards be duly organized with the perfect personnel and with the proper stimulus for active work unless adequate compensation for their services are provided for by the respective city or town?"

I am compelled to state that I believe too many Boards of Health are now in a state of peaceful quiescence, that many are dead and no intelligent post-mortem has been held; that many Boards of Health are still shackled to political affiliations and hence their activities are liable to be curtailed.

Then too, many Boards of Health are crippled in the appropriations, and therefore, through no negligence on their part, their activities are restricted.

Sometimes I am compelled to ask the question, "Are Boards of Health looked upon or considered with that degree of confidence and high standing to which they are entitled?"

Why the manifestation of that spirit of antagonism, so frequently displayed when the activities of the Board are put in execution?

Hence, the question of today is very apropos, "What activities should Boards of Health undertake?"

Foremost of all is, the conservation of health, whether it be the infant, the child, the youth, the adult, or those advanced in years.

It is the imperative duty of every Board of Health to vigorously exert itself in behalf of Preventive Medicine in all its phases.

Naturally, communicable diseases occupy the first place, and the activities of the Boards of Health throughout the state should be so extended that every child may receive the benefit of the Schick and the Dick tests.

I grant that it may be a long and arduous task to convince the people of the advantages of the above mentioned measures and that the activities of Boards of Health will be taxed to the limit, but when diphtheria and scarlet fever are reduced to a minimum or possibly exterminated, then Boards of Health will realize that their activities have been crowned with success.

All communicable diseases as a class, therefore, demand the most vigorous action on the part of the Boards of Health to limit the contagion; to regulate the quarantine; to prevent intermingling of suspicious cases; to supervise transportation; to protect the wage earners; and such activities cannot be shirked.

It is a continuous and ever increasing responsibility thrust on the Boards of Health to maintain their activity at all times amid the varied and numerous matters daily arising.

I would like to see the work of School Inspection brought to a higher degree of perfection and the pupil thus saved from the unknown sources of contagion.

I would like to see more vigorous activity in preventing ophthalmia neonatorum.

I would favor unlimited activity in the matter of food inspection together with close scrutiny of soda fountains and their attendants.

I would demand a regular weekly inspection of all bakeries and the employees engaged therein; and the same activity extended to restaurants and cafes; candy kitchens and ice cream manufactories.

I have advocated posting in all places wheresoever food is served, a Board of Health certificate stating that such place has been duly and regularly inspected by the Board of Health and found to be in excellent sanitary condition, thus assuring the public that the above mentioned place or places meets the approval and endorsement of the Boards of Health, and in so doing I believe the effect for cleanliness would be greatly enhanced.

In every large muncipality the housing question is often a serious problem and it would seem that the activities of the Boards of Health would be more wisely administered by adopting the tenement laws and thus safeguard the health of the community.

Oftentimes the Boards of Health are called upon to abate nuisances both of a major and minor nature, and one of the most frequent to abate is the overflowing of cesspools; this may appear as a trifling mater, but when a person is so inconsiderate of the rights of others or so domineering as to enforce selfish plans so to jeopardize the health and property of others nearby, then the Boards of Health must eradicate such menaces.

It may be a surprise to those present to know that there are no statutes or rules or regulations governing the construction of cesspools. Any man or contractor may construct a cesspool in any way, shape or manner, or in such location as he pleases, irrespective of any future nuisance arising.

I firmly believe that no such cesspool should be constructed without a license or permit from the Boards of Health specifying the location, size and manner of construction.

As every Board of Health owes its allegiance to the state, it can best serve the state and its own community by enforcing the laws of the state, and in so doing merit the respect and confidence of the people at large.

All Boards of Health have mutual responsibilities; mutual interests; mutual aims; mutual endeavors; and a mutual desire to attain the best results.

But why such laxity in the activities of some Boards of Health as to almost subject themselves to be the laughing stock among their confreres; or to so conduct their activities as to stimulate hostile criticism and opposition in the profession?

If the activities were more uniform or more standardized, I believe the power for good would be augmented a hundred fold.

Thanks to the activities of the Boards of Health that typhoid has been practically eliminated; smallpox has been subdued notwithstanding the cry of opposition of the anti-vaccinationists; and incidentally—are all Boards of Health as active as they should be in carrying out the law requiring all children entering the public schools to be vaccinated?

Then again by reason of these activities clean milk is being daily produced; garbage is being properly taken care of; foul toilets and unsanitary plumbing can no longer be allowed to exist by inconsiderate and obstinate landlords; filthy yards are made clean; dumps that are disgusting to the eye and offensive to the smell are converted into plots of grass. How could all these things be accomplished were it not for the activities of the Boards of Health?

But in all the activities of Boards of Health, I am somewhat zealous of the relations between the medical profession and the above mentioned Boards.

Physicians must realize that the Boards of Health are responsible for epidemics; that the individual doctor is responsible for not reporting those cases which the law states must be reported; that he is responsible for an incorrect diagnosis, and if unable to make a diagnosis, he should be required to seek the assistance of the Boards of Health. The time has gone by when every physician can be an expert in every branch of medicine and the sooner the physician digests that fact the better.

Let the physician realize that the Boards of Health in their activities desire to render service and assistance and not to unwarrantably meddle into the private practice of any physician.

By reason of their existence, and their responsibility, Boards of Health must be the dominating factor and exercise the authority vested in them for the protection and health of the community, at large, and in fulfilling their duties, and in so conducting their activities as to merit the confidence, respect and good-will of the public, whose interests they serve.

EXCERPTS FROM THE BULLETIN OF THE AMERICAN SOCIETY FOR THE CONTROL OF CANCER

CANCER CONTROL IN EUROPE—RESULTS OF A RECENT SURVEY MADE BY DR. SOPER

On July 18, 1925, Dr. George A. Soper, the Managing Director of the American Society for the Control of Cancer, sailed for Europe to investigate the subject of cancer control in Europe. He returned October 17, having been gone just three months. He visited England, France, Switzerland, Belgium and Holland. The number of cities visited was 15; the persons interviewed 56; the miles traveled 10,000.

THE LEADING FACTS

Six great facts stand out prominently as a result of this investigation:

First, Europe is thoroughly aroused to the importance of the problems presented by cancer and is taking definite, scientific and practical measures to solve them.

Second, it is the opinion of those who are best informed that the control of cancer can best be accomplished through collective efforts founded on the dual principle of (*a*) research and (*b*) the wide application of all the authentic and useful information which can be obtained about the disease.

Third, the idea that cancer will soon be controllable as a result of some great discovery such as led to the preventive and curative measures which are already in force against certain other diseases, is nowhere entertained.

Fourth, there is no serum or drug in general use for the cure of cancer which has the endorsement of the medical profession in Europe.

Fifth, the procedures which are employed abroad for the diagnosis and cure of cancer are essentially the same as those used by physicians of equal standing in America.

Sixth, in Europe as in America it is recognized that a great many lives can be saved if people will learn the early symptoms of cancer and, when they first think they may have that disease, apply to a competent physician for examination and such treatment as may be necessary under the circumstances.

In England efforts are being directed toward a better understanding of the biological aspects of cancer with the idea that this may lead to the discovery of the cause or causes and eventually to the prevention of the disease. Activity also is shown in the epidemiological side of the question. Both the laboratory research and the epidemiological work are organized and coördinated, and in the hands of persons of good professional ability. The hospital facilities for the care of cancer patients are generally thought to be adequate.

In France a nation-wide effort is being made to promote investigation and provide patients with the skillful care which they need. Centers against cancer containing hospital and research facilities have recently been established in connection with the medical schools which exist under the government.

Switzerland and Belgium are following the lead of France and already possess medical centers supplied with radium, X-ray apparatus and surgical equipment. Holland has long been a leader in research work, and Amsterdam and Groningen have well-established anticancer centers under the direction of men of high scientific attainment.

Under the League of Nations a committee is at work comparing the cancer mortality statistics of different countries, in the hope of shedding light upon the apparent differences in the behavior of the disease among people of various races.

In London, Paris, Geneva, Strasbourg, Brussels and Rotterdam there are radium institutes where patients are treated with ever-increasing skill and the advantages and limitations of this method of treatment are carefully studied.

Research is proceeding chiefly along three main lines. First, in the direction of animal experimentation. The object of this work is to arrive at an understanding of the fundamental phenomena, and particularly the causes, of cancer. Second, along the line of X-ray and radium treatment in the innumerable ways in which these agents may be employed with or without surgery. Third, in the direction of statistical research.

THEORIES AS TO THE CAUSE OF CANCER

An admirable scientific spirit prevails with reference to the way in which the causation of cancer ought to be regarded. Nobody pretends to know all about it or, in fact, very much about it.

That irritation often played a part in the formation of cancer was a nearly universal opinion, but there were cases in which no discoverable form of irritation, physical, chemical or other, had any part in it.

The papers of Gye and Barnard were everywhere known. They were recognized as of stimulating value wherever a laboratory for cancer research or bacteriology existed. Not all the experienced workers who were interviewed, however, were convinced that the whole of the Gye and Barnard work would be corroborated.

TREATMENT

Considerable diversity exists in the method of treatment employed for the cure of cancer. Speaking generally, it may be said that in England preference is usually given to surgery. Some surgeons prescribe little or no radiation. At the Cancer Hospital, London, X-rays have been used very scientifically. At the Middlesex Hospital there is more radium than almost anywhere else in England and a committee on radiation. The Radium Institute, London, has much radium and no X-ray machine. These are all institutions of the best class.

French practise is strongly influenced by the highly scientific Radium Institute of which Dr. Regaud is director. Both radium and X-rays are employed and with a sincere desire to be scientific. The same is true of Belgium. There each center against cancer is equipped with two grams of radium.

SILICOSIS

The Bureau of Mines of the Department of Commerce has recently released for publication the results of investigation into the distribution of silicosis. Silicosis, or miners' phthisis, has been found to be present in many of the states of the United States as well as in New Zealand, Australia, South Africa and Great Britain. In one district 433 miners out of 720 examined had silicosis, and in another 432 out of 1,018.

There are two theories regarding the causation of silicosis, one being that silica dust by its sharp-edged particles injures the lung tissues mechanically, the other that the silica, being slightly soluble in water and weak alkalis, injures lung tissue by its poisonous action.

Generally silicosis is divided into three stages; in the first symptoms and physical signs of damage to the lungs are shown, but capacity for work is not impaired; in the second definite signs of silicosis are present and capacity for work is impaired, although not seriously or permanently; in the third the capacity for work is seriously and permanently impaired.

A cardinal symptom of silicosis is shortness of breath and a cardinal sign is diminished expansion of the chest. Other symptoms and signs such as coughing, frequent colds, pains in the chest, and X-ray findings are important in making a diagnosis. The susceptibility to tuberculosis is increased.

SUBSTITUTE FOR INSULIN

Dr. J. M. Rabinowitch of Montreal, according to *Science*, has prepared a substitute for insulin, dioxyacetone, which may be taken by mouth and causes a lowering of the blood sugar. Dioxyacetone is a sweet syrup, analagous to sugar, which is obtained from glycerine on which a certain bacterium has been allowed to act. It is possible that this may supersede insulin in all but the most serious cases of diabetes, according to present claims.

DISTRICT OF COLUMBIA DRUGGISTS URGE SUPPORT OF VENEREAL DISEASE CONTROL LAW

That druggists should no more attempt to treat the venereal diseases than they should handle smallpox, typhoid, diphtheria, or similar ailments is the view expressed by the Legislative Committee of the District of Columbia Retail Druggists' Association in a circular recently issued by the committee to members of the Association. "Druggists must realize," says the committee, "that efforts of the public health authorities have placed venereal diseases in the same category as smallpox, diphtheria, typhoid, yellow fever and other, more or less controllable diseases." Attention is directed to the venereal disease control law for the District of Columbia enacted by Congress in February, 1925, and the druggists of the District are urged to coöperate with the local health department and physicians in making effective the provisions of the law.—*United States Public Health Service.*

DIPHTHERIA DECREASE

For the ten months of the year ended November first Boston is to be congratulated on the small number of cases and deaths from diphtheria in this city.

While it may be found that the same conditions prevail in other cities, it is none the less noteworthy that for the period mentioned the morbidity and mortality rate from diphtheria in the city has been lower than for any similar period since diphtheria has been a reportable disease. In spite of Boston's increase in population there has been no year since 1861, when statistics are first available, that the actual number of deaths from diphtheria has been as low as during the present year, and with few exceptions, there has been no year since 1871, when cases of diphtheria first began to be reported, that the number of reported cases of diphtheria has been so low. Up to November first, 1073 cases of diphtheria have been reported in Boston with 78 deaths. In the past with a much smaller population, deaths from diphtheria in Boston have been as high as 878 in a single year. The death rate from diphtheria has been decreasing for many years but when it be considered that the average annual number of deaths for the past six years has been 150, and there have been times in the past when over 5000 cases of diphtheria have been reported in a single year, the record for the present year is certainly remarkable. It is evident that the situation with respect to the prevalence of diphtheria in the city this autumn, and up to the present, November 15th, is most exceptional and there is no reason to believe that the totals for the whole year will be found to be materially changed when the figures for November and December are included.

In view of the fact that marked annual fluctuations in cases and deaths from diphtheria have occurred in the past and that the tendency of the death-rate from diphtheria to decrease was apparent even before the discovery of antitoxin, we should not jump at conclusions that our diphtheria problem has been fully solved by present methods of dealing with the disease. Before accepting any such conclusions we must, in the light of past experience, wait until we can see what the death rate from diphtheria in Boston will be for the next two or three years, at least.

The distribution of the deaths from diphtheria with respect to age for the first ten months of this year is shown below:

Under 1 year	1 year	2 years	3 yrs.	4 yrs.
9	18	13	5	9

5 yrs.	6 yrs.	7 yrs.	8 yrs.	9 yrs.	10-14 yrs.	15-19 yrs.	over 20 yrs.
10	4	3	0	2	1	1	3

The number of reported cases and deaths from diphtheria since 1918 is as follows:

Year	Cases	Deaths
1918	2832	217
1919	2670	153
1920	2010	140
1921	2992	148
1922	2992	143
1923	3257	173
1924	2521	168
*1925	1073	78

*Jan. 1 to Nov. 1
10 months

RECENT DEATHS

CADY—Dr. Frederic Benjamin Mooers Cady, for a long time efficient secretary of the Middlesex South District Medical Society, died at the United States Veterans' Hospital No. 89, at Rutland, November 12, 1925.

Dr. Cady was a native of Cooperstown, N. Y., where he was born in 1881. He received an A.B. at Harvard in 1903 and his M.D. at Harvard Medical School in 1907, settled in practice in Cambridge and joined the State medical society in 1911. He practised chiefly neurology and psychiatry. His genial presence will be much missed in his District.

KELLEY—Dr. Walter Henry Kelley, a graduate of Harvard Medical School in the class of 1902, died at his home in Dorchester, November 16, 1925, aged 54.

He was born in South Boston, was a graduate of Boston Latin School, and had practised in Dorchester since graduating in medicine.

He is survived by his widow, who was Ida Mary Fitzpatrick, and by three sons.

SANBORN—Dr. Edwin Aaron Sanborn, a retired Fellow of the Massachusetts Medical Society, died at his home in Somerville, November 14, 1925, at the age of 76, after an illness of several months.

Dr. Sanborn was born at Grafton, N. H., August 28, 1849, and was graduated from Dartmouth Medical School in 1876, whereupon he settled in East Somerville and joined the State society in the same year. Later he moved to Somerville proper and had prac-

tised there since until his retirement in 1915. He is survived by his widow, Mrs. Josephine E. Sanborn.

SAWYER—Dr. Herbert Houston Sawyer, a Fellow of the Massachusetts Medical Society, died at his home in Dorchester, November 13, 1925, aged 55.

He was a graduate of Boston University School of Medicine in the class of 1893, and practised dermatology. He is survived by his widow.

VON GROLL—Dr. Maximilian Charles Von Groll died suddenly at his home in Boston, November 11, 1925.

He was a graduate of Tufts College Medical School in the class of 1898, and then served as house officer at the Carney Hospital, settling in Boston.

He was a member of the Royal Arcanum and was once vice-president of the Catholic Union. He is survived by his widow, who was Celeste T. Cunningham of Columbia, S. C.

OBITUARY

JOHN NEWPORT LANGLEY, 1852-1925, Sc.D., Hon. LL.D., Hon. M.D., F.R.S.

News has recently been received in this country of the death of Dr. Langley, Professor of Physiology in the University of Cambridge. By his death both clinical medicine, and physiology in particular, lose a man of the highest calibre. His research studies, particularly in the field of neurology, threw much light on the vegetative nervous system and added facts of the greatest importance to our scant knowledge of its physiology.

He was trained at Cambridge, England, in the School of Physiology formed by that remarkable personality, Sir Michael Foster. Many of Foster's pupils besides Langley have become illustrious in either clinical medicine or its laboratory branches—Gaskell, Sherrington, Head and Adami, to mention only a few. Langley's earliest papers were on the salivary glands. This experimental work led up to a comprehensive chapter on the subject published in Schäfer's "Textbook of Physiology" (1898). He then turned his attention more especially to the sympathetic nervous system, following and augmenting the work of Gaskell (1886-9), a fellow worker in the same laboratory. Gaskell first made clear the relation of the somatic musculature and glandular system to its nervous supply, and showed that the sympathetic ganglia were connected with the cerebro-spinal system by the white rami. He also demonstrated the gray rami, the medullation of the axones of the sympathetic system in relation to the various ganglia, and the cranial and sacral components of the same system.

Langley continued this research, using the term "autonomic" for the whole system. "The autonomic nervous system means the nervous system of the glands and the involuntary muscles; it governs the 'organic' functions of the body." The results of this fundamental work were published by Langley and Anderson in the *Journal of Physiology,* 1894-6. In Schäfer's "Textbook" (1900) he contributed a paper of 80 pages on "The Sympathetic System." Other important papers on the same subject appeared in *Brain* (1903) and the *Journal of Physiology* (1903). Langley also took up the subject of the voluntary muscles and their innervation, pointing out in all probability, from the functional point of view, that it was not the end-plates of the peripheral nerves that were affected by poisons such as curari and strychnine, but a "receptive substance" contained in the muscle cells themselves—an important contribution to the physiology of muscular movement.

In his later years he showed interest in many aspects of the physiology of the nervous system and did considerable research on non-medullated fibres in peripheral nerves going to skeletal muscles. Other papers dealt with the problem of vascular dilatation of the extremities and its relation to the sympathetic system.

He wrote continuously, usually two or more contributions appearing in the *Journal of Physiology* alone each year, and was actively engaged in research up to the last few months of his life. Practically all his work was done at the Physiological Laboratory in Cambridge. He was a Fellow of Trinity College. As Michael Foster's assistant, he began to edit the *Journal of Physiology* in 1894, became sole editor in 1906-07, and continued in this important office up to the time of his death. His book, "The Autonomic Nervous System," was published by Heffer in Cambridge, England, 1921.

He will long be remembered as a brilliant experimental physiologist who added greatly to our knowledge of the function of the nervous system, especially the so-called autonomic nervous system. Gaskell before him laid down a fundamental conception of the voluntary nervous system, but Langley contributed all the details which made secure the principles already presented by Gaskell's work. He did many other important pieces of research, but none will be so long remembered as his work on the autonomic system.

"The natural desire of good men is knowledge."

Henry R. Viets.

CORRESPONDENCE

THE CALCUTTA SCHOOL OF TROPICAL MEDICINE

Research in Indian Diseases—Results of Past Investigations

(From a Correspondent)

The Calcutta School of Tropical Medicine is one of the great institutions of the world. It owes its origin to the efforts of Sir Leonard Rogers, whose discoveries in cholera, amoebic dysentery, kala azar, and leprosy are well known. It was in 1910 that the idea of a school of tropical medicine in India was

first conceived by him, and, before he retired in 1920, the school came to be an accomplished fact, a deserving monument to a great benefactor of mankind. The aim of the school has been to make it a centre for research work for the whole of India as well as a teaching institute in which selected medical men from every province will obtain instruction which will raise the standards of medical education and public health throughout the country. A brief survey of the activities of the school is alone possible to be presented here, which, it is hoped, would help to a realization of the work that is carried on.

FIELDS OF RESEARCH

The field of scientific endeavor to which the school has given attention is very extensive. Principal among diseases investigated are hook-worm disease, kala azar, leprosy and diabetes. In hook-worm disease, Dr. A. C. Chandler has investigated various phases and has taken advantage of opportunities offered for the study of other worms, parasites of man and animal. Much has been done in the treatment of disease caused by worms, particularly as regards investigating the effectiveness and safety of carbon tetrachloride for hook-worm and thread-worm infections. Studies have also been made of the spread of hook-worm eggs by domestic animals, the migration and habits of hook-worm larvae in soil, and the value of egg counts in the estimation of the degree of infection of communities.

The research in kala azar is under the direction of Dr. Napier, best known to the medical world by the very valuable simple blood test which he introduced for the diagnosis of the disease. Dr. Napier has been responsible for testing and bringing into every day use two very valuable drugs for the treatment of the disease. He along with Major Knowles and Dr. Smith is now engaged in determined efforts to detect the insect which is responsible for conveying kala azar to man. They have brought forward evidence which throws the gravest suspicion on a kind of sand fly; should this prove to be the carrier of kala azar, the last of the really great problems of tropical medicines will have been solved.

LEPROPSY, DIABETES, CHOLERA

The Leprosy Research Department is financed chiefly by the Indian Research Fund Association. Dr. Muir has continued his well-known work on the methods of treatment of leprosy, and has made further progress in the discovery of simpler and more effective treatment. It is becoming increasingly clear that leprosy is far from being an incurable disease; if taken in time it is singularly amenable to treatment; and if patients can be encouraged to come early, they will not become sources of danger to the community. The out-patients' department at the Calcutta School is now accepted as the pattern on which it will be possible to devise treatment centres for lepers. No less than twenty medical men have attended the laboratory and been instructed in the modern methods of dealing with this disease, which at last has ceased to be the despair of medicine. His Excellency the Viceroy has recently issued an appeal for funds for the control of this fell disease.

Of special interest is the Diabetes Research Department, financed by the generosity of an Indian lady—Mrs. Mitra—in memory of Dr. Mitra, a distinguished medical man who, in later life, became Minister in Kashmir.

Dr. J. P. Bose is especially interested in the prediabetic stage, during which the foundations of the disease are being laid. He has confirmed the opinion that the condition which leads to diabetes can be counteracted most effectually by exercise, and that lack of exercise combined with a diet too rich in carbohydrates forms the conditions which are followed by diabetes.

As regards cholera, it may be mentioned here that Captain Maitra's investigations of the conditions among laborers in the Jheria coal-fields led to considerable progress in the important subject of distinguishing true cholera from cases of other diseases which resemble cholera.

OTHER INVESTIGATIONS

Colonel Megaw, who is professor of tropical medicine and also the Director of the School, has studied several diseases from the clinical side. He has gradually accumulated convincing evidence that there is a fever in India which resembles typhus fever, but which attacks persons living in close association with the life of the jungle. To this disease he has given the name "Tick Typhus" because the disease is believed to be conveyed by a tick and because of its clinical resemblance to typhus fever.

Colonel Megaw has also been deeply interested in epidemic dropsy, and for the past fifteen years he has been convinced that it is caused by a poison which develops in rice under certain conditions of manufacture and storage. He calls attention to the necessity for close study of the methods of dealing with rice, which is the staple article of diet of millions of people in the East.

Major Knowles and Dr. Das Gupta have been working at a number of problems. Experimental trials are on hand of several new drugs in the treatment of amoebic dysentery—such as *Kurchi*, an indigenous Indian drug, yatren and stovarsol.

Major Chopra's work has aroused great interest throughout India. He has studied a considerable number of drugs which have a reputation for efficacy among the practitioners of the indigenous system of medicine. These can now be used by medical men with confidence as their properties, and doses have been worked out with scientific accuracy. The work is not merely of medical interest, it is also of economic importance; drugs of Indian origin have been found to be just as effective as those imported from abroad and also much cheaper. It is also found that there are plants which grow in India; though not identical with European medicinal plants. These may have very similar properties and so can serve as useful and inexpensive substitutes.

SOME RECENT DEVELOPMENTS

Among the recent developments of the school must be mentioned the opening of the Pasteur Institute in June, 1924. The school was fortunate in securing the services of Dr. E. C. R. Fox, who has an unrivalled experience of antirabic work in India. Quickly it leapt into popularity and the total number of patients attending from June 1 to December 3, 1924, was about 2,000.

Another development has been the Institute of Hygiene. The staff in this Institute is said to be unequalled in the East and facilities have thereby been provided for giving a thorough course of instruction in public health to medical men in India. It is obvious that a staff like this working in India deals with the subjects of instruction in a manner which has a far more direct application to the public health problems of India than is possible in any institution in Europe.

Instructions in certain specialities will still have to be sought in Europe and America, but systematic training in public health can be obtained in Calcutta such as is not available elsewhere in India or in the whole world.

A disease survey of India is now in course of preparation. This when complete will form a more accurate index of the distribution of the great diseases of India than has hitherto been available. It is also proposed to provide a properly equipped museum of public health.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

Diseases Reported for the Week Ending November 14, 1925

Anterior poliomyelitis	3	Pneumonia, lobar	130
Chickenpox	225	Scarlet fever	180
Diphtheria	103	Septic sore throat	3
Dog-bite requiring anti-rabic treatment	2	Syphilis	67
Encephalitis lethargica	2	Suppurative conjunctivitis	6
German measles	21	Trichinosis	1
Gonorrhea	182	Tuberculosis, pulmonary	112
Hookworm	1	Tuberculosis, other forms	10
Influenza	5	Tuberculosis, hilum	19
Measles	761	Typhoid fever	11
Mumps	55	Whooping cough	204
Ophthalmia neonatorum	18		

RHODE ISLAND STATE BOARD OF HEALTH

Contagious Diseases During the Period from Week Ending October 17, 1925, to November 7, 1925

Cerebrospinal meningitis	1	Ophthalmia neonatorum	6
Chickenpox	22	Paratyphoid fever	1
Diphtheria	35	Pneumonia	5
Encephalitis	1	Poliomyelitis	4
German measles	1	Scarlet fever	32
Influenza	18	Smallpox	11
Measles	125	Tuberculosis	28
Mumps	7	Typhoid fever	5
		Whooping cough	35

NOTICE

The Massachusetts General Hospital and The Boston City Hospital will notify the Boston Medical and Surgical Journal of surgical operations listed each day.

This information will be transmitted to all who may apply by telephone at 9.05 a. m. or later.

REPORTS AND NOTICES OF MEETINGS

SPECIAL MEETING

A special meeting of the Boston Orthopedic Club in combination with the Boston Surgical Society will be held Thursday evening, December 3rd, in the Aesculapian Room, Harvard Club, at 8:15 p. m. Program:

Fascia in the repair of Anatomical defect, W. Edward Gallie, Assistant Professor of Surgery, University of Toronto, Faculty of Medicine, Surgeon-in-Chief, Hospital for Sick Children.

P. D. Wilson, *Secretary.*

HARVARD MEDICAL SOCIETY

It is expected that Prof. V. Putti will lecture before the Harvard Medical Society at the Peter Bent Brigham Hospital, Monday evening, November 30, at 8:15 on "The History of the University of Bologna."

THE FIFTH WEEKLY MEETING OF THE PHYSIOLOGICAL CONFERENCES

The fifth weekly meeting of the Physiological Conferences will be held in the Bowditch Library of the Harvard Medical School on Monday afternoon, November 30, at 4 o'clock. Dr. E. S. A. Robinson, Assistant Director of the Massachusetts Antitoxin and Vaccine Laboratory, will speak on "The Combination and Dissociation of Toxin and Antitoxin Mixtures."

MEETING OF THE TRUDEAU SOCIETY

The Trudeau Society of Boston will meet at The Boston Medical Library, December 8, 1925, at 8.15 P. M.

Speaker, Dr. John B. Hawes, 2nd. Subject, Hilum Tuberculosis. Dr. R. D. Leonard, Dr. H. D. Chadwick, Dr. F. H. Hunt and Dr. G. W. Holmes have been asked to become parties to the discussion.

Members of the Medical Profession are cordially invited to attend this meeting.

George S. Hill, *Secretary.*

BERKSHIRE SAFETY COUNCIL

At 8 p. m. on Thursday, December 10th, in the auditorium of the Pittsfield Electric Company on Eagle Street, Pittsfield, the Berkshire Safety Council will hold a meeting at which there will be a demonstration of resuscitation from electric shock. There will be a general discussion of methods of resuscitation.

All members of the medical and dental profession are invited to attend and take part in the discussion.

BERKSHIRE DISTRICT MEDICAL SOCIETY

The Berkshire District Medical Society will hold a regular meeting in the rooms of the Park Club, Pittsfield, on Friday, November 27, 1925, at 8 P. M.

The North Berkshire Medical Society and the Berkshire Dental Society are invited to attend the dinner and meeting.

The subject of anesthesia in its various forms will be thoroughly discussed and illustrated by some of the very highest authorities on the subject, and there will be opportunity for questions and thorough discussion by our members and friends.

James T. Gwathmey, M.D., of New York City, will explain "Synergism and the Newer Anesthetics."

W. Wayne Babcock, M.D., Surgeon-in-Chief of Samaritan Hospital of Philadelphia, will discuss "Spinal Anesthesia."

Gaston Lavat, M.D., of New York City, formerly lecturer on "Regional Anesthesia" at the Mayo Clinic, has chosen as his subject, "Practical Value of Regional Anesthesia in General Surgery."

A lobster dinner will be served in the dining room of the Park Club at 6.30 p. m.

HAMPSHIRE DISTRICT MEDICAL SOCIETY

THIS Society met Wednesday, November 11, at Forbes Library, Northampton.

PROGRAM

1. Dementia Praecox—Dr. Houston. Discussion opened by Dr. Pierce.
2. Surgical Notes—Dr. Perry. Discussion opened by Dr. C. T. Cobb.
3. Tonsils—Dr. Young. Discussion opened by Dr. Shores.
4. Diabetes—Dr. Smith. Discussion opened by Dr. Copeland.

THE WORCESTER DISTRICT MEDICAL SOCIETY

THE November meeting of the Worcester District was held at the Worcester State Hospital on November 11, 1925. At 5.30 in the afternoon there was held a short business meeting with President Royal P. Watkins presiding.

At 6.30 Dr. Bryan invited the members to a bountiful repast. The dining hall was attractively decorated, the waitresses were courteous and obliging (nurses) and the food was tastily prepared and served. This Society is always pleased when Dr. Bryan entertains.

Following the supper, Dr. Watkins introduced the speaker of the evening, Dr. Warren Stearns of Boston, psychiatrist to the Charlestown State Prison. Dr. Stearns' subject was "Conduct Disorders as a Public Health Problem." Dr. Stearns stated that the three main factors which guided one's conduct were: (1) The Church, (2) Law, (3) Public Opinion. He then classified those that more commonly suffered from a conduct disorder. The class that made up the most of these disorders were the children who during the period of adolescence could not seem to master themselves. Some of these children at maturity became law-abiding people. The feeble-minded made up another group. A third class included the insane. Many of us doubted if there were any insane people after hearing Dr. Stearns discuss this group. Another class were the psychoneurotics. A fifth group that suffered from conduct disorders were the abnormal personalities. Still another group were those with physical handicaps. Drug and alcoholic addicts were very apt to have conduct disorders. Wealthy seniles and also some poor ones made up another group. Environmental reactions play an important factor in these disorders. Dr. Stearns concluded his talk by answering the questions of those present. This talk was one of the best that this Society has listened to for some time. Dr. Stearns held our attention constantly and we all felt that we would like to know this man better if time had permitted. It is hoped that we may again be privileged to hear this gentleman from Boston at some future meeting.

The meeting was adjourned at 9 o'clock.

The next meeting of this Society will be held at the Boston Store Restaurant on Wednesday, December 9, 1925.

Dr. Morton Langille has become associated with Dr. E. E. Smith in the practice of X-ray and radium.

EARL E. FIPPHEN.

MEETING OF HARVARD MEDICAL SOCIETY

THE Harvard Medical Society met at the Peter Bent Brigham Hospital on Tuesday evening, Nov. 10th. Two cases were presented. The first was a woman aged 32 who came in because of transient loss of speech and transient paralysis of the left arm. In 1920 she had a severe attack of influenza, just at the beginning of her first pregnancy. She had fainting spells about every four weeks until the end of the fifth month. After delivery, the attacks returned and she was put to bed until they disappeared. The transient loss of speech and paralysis of the left arm began this year on Oct. 2nd. Physical examination showed signs of mitral stenosis and an enlarged heart. A little later she had an attack of blanching of the leg. A similar second attack came four hours later and lasted three hours. With this there was numbness and severe abdominal pain. The symptoms gradually disappeared. The problem in this case is to explain the sudden disturbance, evidently vascular and its equally sudden disappearance.

The second case was a man, 33 years of age, who first appeared in 1923 with a question of right kidney stone. Examination proved negative. He improved but still had pain in the right side. Culture from right ureter was taken and B. Coli found. On second entry, he complained of a scatchy feeling in the right side. The findings were negative. On the third entry, in September of this year he had the same complaint. A pyelolithotomy was done and a small uric acid stone removed from the right pelvis. After a stormy convalescence, the patient was discharged practically well on Oct. 6th. A little later he returned with a complaint of pain accompanying respiratory movements and also pain in the right flank. X-ray show areas of decreased density in the thorax. It was assumed that he had a small sub-diaphragmatic abscess with evolution of gas.

Dr. Davidoff gave an interesting account of his experiences with the MacMillan expedition to the Arctic regions this summer. At one point in Greenland, an epidemic of whooping cough was found among the natives. Immunity to the disease apparently had not been developed. The old men were whooping as well as the children. Twenty per cent of the cases died of broncho-pneumonia. The doctor there was using serum as a prophylactic but was discouraged about it. He will write about it later. Dr. Davidoff believes that it is a good clinical experiment.

On Aug. 1st, in a blinding snow storm and through great herds of walrus, they landed at Etah and stayed a month. There was no alteration between day and night. The Esquimaux sleep when they feel tired and sleep till they are rested; then they stay up until they are tired again.

Their food consists largely of protein material with some fat. Dr. Davidoff took the blood pressure of all the inhabitants. The blood pressure of the oldest resident was 120 systolic and 60 diastolic, and that of the others corresponded. There was nothing suggesting low kidney function.

Diseases among the Esquimaux are few. There is no tuberculosis. Apparently they would live forever, but that some freeze to death, some are drowned and a few starve.

Treatment of the native medicine man consists of noisy incantations. They do have women trained as mid-wives who are equal to some acting in that capacity here. The infants are breast fed. If the mother dies and there is no wet nurse, the infant dies too. Sometimes the child is nursed until six or seven years old. Mr. MacMillan tells of a child of six, standing between his mother's knees, refreshing himself between puffs on his pipe. The story of the late maturation of the girls is correct. They begin to menstruate at about twenty, although they are married at the age of thirteen or thereabouts.

Dr. Davidoff's story of his experiences in the Arctic was illustrated by lantern slides from pictures taken on the trip.

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SOCIETY MEETINGS

DISTRICT MEDICAL SOCIETIES

Essex North District Medical Society

January 6, 1926—The semi-annual meeting at Haverhill.
May 5, 1926—The annual meeting at Lawrence.

Middlesex East District Society

January 13—At the Harvard Club at 6:30 P. M. Address by Dr. Richard Ohler, "Metabolism."

February 10—At the Harvard Club. Address by Dr. William F. Boos; subject, "Industrial Poisoning."

April 14—At the Harvard Club at 6:30 P. M. Address by Dr. William E. Ladd, subject to be announced later.

May—Annual meeting, Colonial Inn, North Reading. Subject and speaker to be announced.

Suffolk District Medical Society

January 6—At 8:15 P. M. Medical Section (meeting postponed from December). Dr. W. J. MacDonald will speak on "Experimental Work in High Blood Pressure."

January 27—At 8:15 P. M. Combined meeting with Boston Medical Library. "Medical Experience of an Explorer," Dr. J. Hamilton Rice.

February 24—At 8:15 P. M. Surgical Section. "Post-operative Care of Surgical Cases," Dr. Dean Lewis, Chicago. "Surgical Convalescence," by Dr. John Bryant.

March 31—At 8:15 P. M. Medical Section. Subject to be announced later.

April 28—At 8:15 P. M. Annual meeting. Election of officers. "Some Diagnostic, Prognostic and Therapeutic Aspects of Disorders of the Blood," Drs. George R. Minot, Cyrus C. Sturgis and Raphael Isaacs.

Notices of meetings must reach the JOURNAL office on the Friday preceding the date of issue in which they are to appear.

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BOOK REVIEW

Diet in Health and Disease. By JULIUS FRIEDENWALD, M.D., Professor of Gastro-Enterology in the University of Maryland School of Medicine, Baltimore; and JOHN RUHRAH, M. D., Professor of Diseases of Children in the University of Maryland, Baltimore. Sixth edition, thoroughly revised. Cloth, $8.00 net. Octavo of 987 pages. Philadelphia: W. B. Saunders Company. 1925.

The latest edition of this valuable standard reference work on diet, appears in formidably encyclopedic size, an indication to this extent of the degree of success which the authors have attained in their attempt "as far as it is possible in one volume, to bring together the greatest possible number of facts relating to foods and nutrition in health and disease."

"The aim of the book is entirely practical," as was that of the first edition, which however also aimed to be "concise" and "simple enough to be used for rapid reference by the busy practitioner."

The busy practitioner may find himself well satisfied; he may however find himself questioning if this latest edition has not so widened its scope as to make difficult an agreement with the title-page injunction from Burton to the effect that "These few rules of diet he that keeps, shall surely find great ease and speedy remedy by it."

Burton doubtless would have derived satisfaction from the fact that in an American text book of 1925 vintage, 12 pages (nearly one-half of the space assigned to Beverages and Stimulants) are given over to a consideration of the uses of Alcohol.

Burton doubtless also would have derived satisfaction from the services of a coryphaeus competent to guide him through the maze of 500 or more pages of laboratory and scientific and miscellaneous information, to the 400 or less pages upon Diet in Disease, wherein, should he be in quest of them, he would eventually discover the less than two pages out of a total of 987, with which the authors dismiss in this "entirely practical" book, such an ever present and ever difficult problem as Diet in Chronic Intestinal Stasis.